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THE PLANT DISEASE REPORTER

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SOME EXPERIMENTS CONCERNED WITH THE FORMATION AND INHIBITION OF APOTHECIA OF SCLEROTINIA SCLEROTIORUM (LIB.) D BY.

D. M. Mc Lean[1]

Abstract

Infections of cruciferous seed plants by Sclerotinia sclerotiorum are incited by ascospores at the loci of deposition of dead flower parts following pollination. Apothecia developed from sclerotia placed in moist sand, soil, sphagnum, and 1% water agar. Apothecia developed in full light, but only stipes developed in amber and black-painted bottles. Sclerotia buried in soil at 6- and 8-inch depths were viable and produced apthecia after 5 years. Disintegration of sclerotia was more rapid in moist sand than in moist soil and nearer the soil surface. Only partial destruction of sclerotia resulted from burning crop refuse after seed harvest. A number of chemicals were tested for inhibition of apothecia when they were applied to the soil over implanted sclerotia. Only calcium cyanamid and Mylone completely controlled this stage of growth. Terraclor and Vapam also were highly effective.

INTRODUCTION

Sclerotinia sclerotiorum (Lib.) D By. causes a serious stalk rot of cruciferous seed plants in the Puget Sound region of western Washington (6). The fungus is widespread in the seed-producing area of the Skagit Valley. Infections are incited by ascospores at the loci of deposition of dead flower parts following pollination (6). Seed stalks are invaded through axillary infections. Frequent precipitation during the pollination period promotes apothecial formation and causes blossom parts to adhere to the foliage and thus establish infection courts. Infections are rarely observed on young cruciferous plants, and cabbage transplants set in infested soil are seldom invaded from the soil. Fungicides applied to the foliage of seed plants by airplane have not given adequate control. It was demonstrated earlier that stalk infections can be avoided by manually removing the cauline leaves before the blossoms fall (7).

Certain studies have been carried out for several years on some of the environmental factors conducive to the formation of apothecia and ascospores and chemical treatments that might inhibit the development of these structures. This report summarizes briefly results from these studies.

PROCEDURE AND RESULTS

Development of Mature Apothecia

The method most commonly used for the production of apothecia from species of Sclerotinia consists of embedding mature sclerotia in moist sand or soil, but other substrata have been used (1, 3, 5, 10, 11). Constant moisture, moderate temperature and relatively long exposures are important in the initiation of apothecia (3, 5, 10, 11). Light is not necessary for the initiation of apothecia, but apparently is necessary for the production of mature apothecia (3, 5, 11).

Mature sclerotia from infected plant tissues were embedded in the following media placed in 16-ounce stoppered bottles: moist sand, soil, sphagnum, and 1% water agar. The bottles were placed in a greenhouse where diurnal temperatures fluctuated from 14° to 20° C. After 6 to 8 weeks, or longer, mature apothecia developed in all media, the greatest percentage in 1% water agar.

Sclerotia were embedded in moist sand placed 1) in amber-colored bottles, 2) black-painted bottles, and 3) clear bottles tightly stoppered and placed in the same environment as

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described previously. Mature apothecia developed normally in full light. Only stipes without hymenium developed in the amber and black-painted bottles.

Longevity of Sclerotia in Soil

It is reported that sclerotia germinated readily and produced ascospores after being held in dry storage for 7 years (4). Several workers (12) reported that sclerotia may remain in a resting stage as long as 10 years and still retain their viability.

Approximately 300 sclerotia of S. sclerotiorum, obtained from cabbage seed cleanings during the milling process, were placed between layers of 10-inch square plastic (lumite) screen bound together with wire staples. The enclosed sclerotia were buried at depths of 2, 4, 6, and 8 inches in three separate areas: 1) sand, 2) well-drained silt loam, and 3) poorly drained soil. At intervals of 6 months over a period of 5 years, sclerotia were removed from each depth at each location and exposed to well-moistened soil for apothecial formation. None of the sclerotia produced apothecia in place.

The sclerotia nearer the soil surface showed the most rapid disintegration. Those in sand decomposed more rapidly than those in soil. Sclerotia placed in poorly drained soil were under water for a week or longer during two separate winters, but remained viable and produced apothecia. Moore (8) reported that in Florida complete decay of sclerotia resulted within 23 to 45 days of continuous or discontinuous flooding in marl, muck, or sandy soils regardless of the depth of burial. In the present experiment sclerotia from the 6- and 8-inch depths produced apothecia after 5 years; however, destruction was nearly complete after this length of exposure.

Destruction of Sclerotia by Burning Seed Crop Refuse

Combines used for seed harvest deposit plant materials on the soil after separating the seed. Where stalks are severely infected by Sclerotinia, many larger sclerotia pass through the combine and smaller ones are separated with the seed. A common practice is to burn the "straw" after harvest. This procedure has been suggested as a practical method of destroying sclerotia (9).

To determine the relative percentage of sclerotia that might be destroyed in this manner, sclerotia were placed between layers of enameled wire screen bound together with wire staples and scattered promiscuously in the crop debris behind the combine. After the usual burning operation, the sclerotia were collected and tested for viability. Only about 40 percent were destroyed by burning. Certain sclerotia were unaffected apparently because they were not exposed to flame.

Chemical Inhibition of Apothecia

Brants (2) states that formalin was more effective than thiram, brassicol, or mercuric chloride for destroying sclerotia of Sclerotinia minor. Germination was inhibited also after 1 1/4 hours at 70° C. Steam sterilization of the soil is reported to give good results in practice (2).

Studies were carried out to determine the inhibition of apothecia by chemicals applied to the soil. During April, approximately 200 sclerotia were distributed on the soil surface in each of a series of greenhouse flats. Sclerotia were lightly covered with soil and well watered, and the flats were placed out-of-doors where they were exposed to natural precipitation. After 3 weeks the soil surface over the sclerotia in separate flats was given the following treatments in 6 replications: dinitro amine (triethanol, isoproponal salt of dinitro ortho secondary butylphenol), 2 and 4 quarts per acre; dinitro general (4,6-dinitro ortho secondary butylphenol), 2 and 3 quarts per acre; monuron (3-(p-chlorophenyl)-1,1-dimethylurea), 1 and 2 pounds per acre; and calcium cyanamid, 300 pounds per acre. Untreated flats provided controls. The failure of apothecia to develop subsequent to the treatments was an index to the inhibition of growth. Only calcium cyanamid completely inhibited apothecial formation and caused rapid disintegration of the surface sclerotia. Results from other treatments were not conclusive because the soil dried within the flats.

During several winters, related experiments were carried out in the greenhouse where temperatures ranged from 15° to 20° C. Square-foot units of soil in benches were separated with wood lath. Sclerotia were distributed on the soil surface at the rate of approximately 150 per unit, lightly covered with soil, and kept well moistened during the course of the ex-

periments. After 3 weeks, and before apothecia had developed, various chemicals were applied to the soil surface over the sclerotia. Apothecia that developed on soil given the different treatments were counted. In addition to the materials mentioned previously, the following chemicals were tested: diuron (3-(3,4-dichlorophenyl)-1,1-dimethylurea), 1 and 2 pounds per acre; Vapam (sodium methyl-dithiocarbamate), 1/2, 1 and 2 quarts per 100 square feet; Dowicide G (sodium pentachlorophenate), 100 and 200 pounds per acre; Terraclor (pentachloronitrobenzene), 30, 100 and 200 pounds per acre; and Mylone (3,5-dimethyltetrahydro-1, 3-5, 2H-thiadiazine-2-thione), 150, 300 and 500 pounds per acre.

Calcium cyanamid and Mylone at each of the rates tested completely inhibited apothecial formation. Terraclor and Vapam in that order were highly effective, but several apothecia did develop even at the higher rates of application. From 13 to 80 apothecia developed in untreated checks in several experiments. Experiments with certain soil sterilants and other chemicals are being continued in greenhouse and field tests.

Calcium cyanamid (1000 pounds per acre granular), Terraclor (15 pounds per acre emulsifiable), and Terraclor (20 pounds per acre of 20% dust) were applied to the soil in 1-acre blocks in cabbage (Brassica oleracea var. capitata) seed fields during June 1957. Applications were made between the rows and as close to the plants as possible after the last cultivation and before pollination. Owing to little precipitation and dryness of the soil, only a few infections occurred in the untreated sections and the results are inconclusive. Calcium cyanamid gave excellent weed control but delayed maturation of seed plants approximately 1 week. Calcium cyanamid at 900 pounds per acre delayed maturation of beans in Wyoming(12).

DISCUSSION

A single apothecium gives rise to a great number of ascospores. It is difficult to make thorough and uniform applications of chemicals to the soil around seed plants. The question arises as to what degree of disease control, if any, might be expected within the treated area if only partial control of apothecia resulted. It is not known how far ascospores can be dispersed in air currents from other sources. It has not seemed feasible to test chemicals in pre-planting soil treatments with biennial seed plants because such treatments would have to be residual during the winter and remain effective after several spring cultivations.

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EFFECT OF FREEZING ON VIABILITY OF THE LIMA BEAN DOWNY MILDEW FUNGUS (PHYTOPHTHORA PHASEOLI THAXT.)

R. E. Wester, Charles Drechsler, and Hans Jorgensen[1]

The lima bean downy mildew fungus (Phytophthora phaseoli Thaxt.) has been kept at Beltsville, Maryland, in a viable condition at -10° F temperature for over 100 days without any reduction in pathogenicity.

In the past it has been difficult to maintain this fungus for any considerable period in the laboratory. In the greenhouse the organism has been kept viable during fall, winter, and spring by inoculating a new lot of lima bean seedlings every 10 days with spores from previously infected seedlings. This procedure requires much time and some facilities for maintaining proper humidity and temperature. During the summer the fungus dies because of excessively high temperatures. The freezing method described here removes these hazards and makes the storing of the downy mildew fungus until required for use a simple matter.

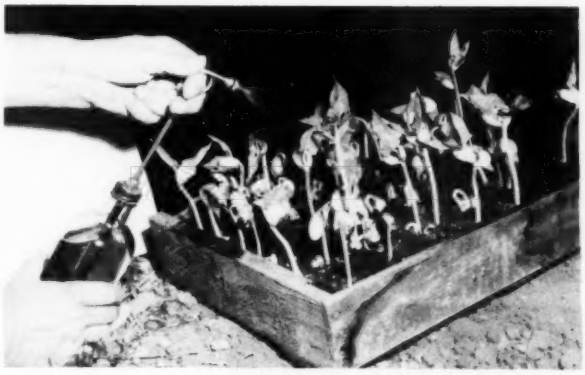

FIGURE 1. Inoculating lima bean seedlings with a spore-suspension of downy mildew fungus.

FIGURE 2. Downy-mildew-inoculated lima bean seedlings being placed in the humidity chamber.

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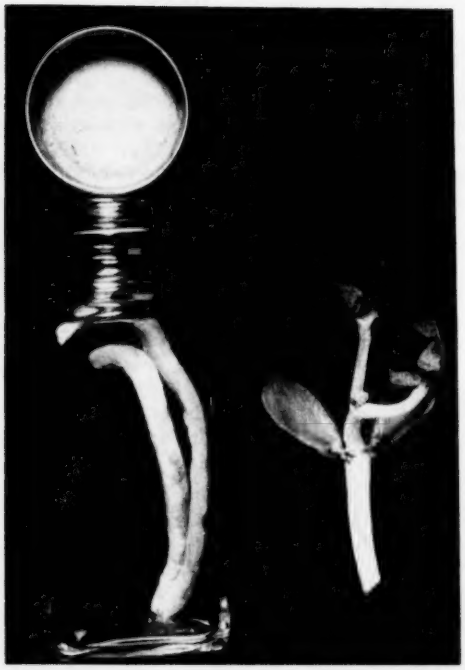

FIGURE 3. Short pieces of downy-mildew-infected lima bean stems in 2-1/2-ounce screw-top jar for storage in refrigerator at -10° F.

FIGURE 4. A flat of lima bean seedlings, 5 days after inoculation with the downy mildew fungus from infected seedling pieces that had been stored in a refrigerator for over 100 days at -10° F.

The method of preparing, freezing and storing the sporulating downy mildew fungus is as follows: Lima bean seedlings emerging from soil in a flat are sprayed with a water suspension of downy mildew conidia (Fig. 1) and placed in a saturated humidity chamber (Fig. 2), in which the temperature ranges from 65° to 75° F. After they have been kept 4 days in this chamber, the seedlings usually become overgrown rather extensively with young conidiophores, which form a downy turf visible to the naked eye. The flat is then transferred to a neighboring 50 percent humidity chamber where within 24 hours further growth of conidiophores usually takes place and conidia are produced in enormous quantity. The seedlings with sporulating conidiophores are cut into short pieces, which are placed in a 2-1/2-ounce

bottle with screw top (Fig. 3) and stored in a -10° F refrigerator.

When required for use, the seedling pieces are placed in an Erlenmeyer flask with water immediately upon removal from the refrigerator and shaken for about 30 seconds. By this treatment the conidia are dislodged from the tangle of conidiophores and set free in the water. The water with the conidia or sporangia in suspension is then atomized on a new stand of lima bean seedlings (Fig. 1). Figure 4 shows a close-up of lima bean seedlings infected with the downy mildew fungus that had been stored for over 100 days at -10° F.

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EXPERIMENTS ON THE CONTROL OF DOWNY MILDEW OF BROCCOLI AND BACTERIAL SPOT OF LIMA BEAN WITH STREPTOMYCIN[1]

Jack Altman[2] and B. H. Davis

Field experiments were conducted during the summer of 1956 with streptomycin for the control of downy mildew (Peronospora parasitica) of broccoli and bacterial leaf spot (Pseudomonas syringae) of lima beans. Applications of streptomycin were applied after the diseases were present in the field in an effort to determine the value of such sprays in reducing these diseases. Since both diseases are sporadic in their occurrence, a regular protective spray schedule is not likely to be profitable in most years. Applications made after the appearance of the disease, if effective, would be of value in the control of these diseases.

DOWNY MILDEW OF BROCCOLI

A severe outbreak of downy mildew of Calabrese broccoli in central New Jersey was reported in July, 1956. Although a 10-day dusting schedule, using captan and Spergon, was being used, satisfactory control of the disease was not obtained. An experiment was set up using antibiotic sprays to control the disease.

Five sprays of streptomycin nitrate (Phytomycin) at 100 ppm and 50 ppm, with and without 1% glycerine were applied at approximately 7-day intervals to replicated 20-plant plots. The sprays were applied with a knapsack sprayer on July 18, 25, August 1, 10, and 15. Leaf samples were collected from both the upper and lower parts of the plants on August 31 and graded according to disease severity. Data are reported in Table 1.

Five sprays of streptomycin nitrate at 50 ppm and 100 ppm, with and without 1% glycerine, significantly reduced downy mildew infection. Sprays of 100 ppm streptomycin were better than sprays of 50 ppm with and without glycerine and streptomycin sprays at both concentrations were slightly better with glycerine than when used alone.

Observations of several adjacent broccoli plantings dusted regularly with captan or Spergon indicated a disease rating of medium to severe on all plants examined, with the greater degree of severity on the lower leaves. Seedlings in nearby beds were also severely infected.

In the same field where the aforenamed test was conducted an earlier spray test using 400 and 200 ppm streptomycin nitrate, with and without 1% glycerine, resulted in considerable injury. The sprays were applied on July 11, 1956, and injury was noted 1 week later. The injury resulted in an overall bleaching or yellowing and a severe stunting. No further sprays were applied but the plants were observed weekly thereafter. On August 10, 1 month after spraying, the injured plants had produced normal new leaves and showed some semblance of recovery, but the bleached condition of the older leaves was still evident.

Sprays of 100 and 50 ppm streptomycin, with and without glycerine, used in the test for which data are given in Table 1, also resulted in slight mosaic-like symptoms. The injury was greater at 100 ppm than at 50 ppm and greater with streptomycin plus 1% glycerine than with streptomycin used alone. This injury due to streptomycin has been observed by the senior author on other crops during the past 3 years[3].

The above data indicate that streptomycin nitrate at 50 and 100 ppm, used alone or in combination with 1% glycerine applied after the disease was present in the field, significantly reduced downy mildew of broccoli. Streptomycin at concentrations of 100 ppm and higher resulted in injury to broccoli foliage, but when sprays were discontinued the plants eventually recovered.

Although a significant reduction in disease severity was obtained with five applications of streptomycin, the cost and the possibility of slight injury to plants make the program of questionable value at this time. However, spraying of broccoli seedlings in the plant bed with a 200 ppm streptomycin spray prior to transplanting may perhaps result in better and more economical control of downy mildew and if injury does occur, recovery in seedlings may be more rapid than in older plants.

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[1] Paper of the Journal Series, New Jersey Agricultural Experiment Station, Rutgers University, the State University of New Jersey, Department of Plant Pathology, New Brunswick, New Jersey.

[2] Colorado State University, Fort Collins, Colorado; formerly fellow, Department of Plant Pathology, Rutgers University, New Brunswick, New Jersey.

[3] Altman, Jack, and Stephen Bachelder. Susceptibility of some ornamental and vegetable plants to streptomycin injury. Plant Dis. Reptr. 40: 1081-1083. 1956.

Table 1. Field studies on control of Downy Mildew of broccoli with streptomycin nitrate (Phytomycin) sprays with and without glycerine[a].

Treatment	Leaves showing various degrees of disease severity[b] — Upper Leaves: None %	Trace %	Medium %	Severe %	Mean Disease Rating[c]	Lower Leaves: None %	Trace %	Medium %	Severe %	Mean Disease Rating[c]
Check	16	48	22	14	1.35	8	36	30	26	1.73
Glycerine 1%	10	44	24	22	1.53	2	38	40	20	1.79
Streptomycin nitrate 50 ppm	28	54	14	4	.95**	8	48	30	14	1.48*
Streptomycin nitrate 50 ppm + 1% glycerine	42	46	8	4	.74**	8	68	16	8	1.27**
Streptomycin nitrate 100 ppm	48	48	4	-	.55**	12	58	22	8	1.26**
Streptomycin nitrate 100 ppm + 1% glycerine	52	46	2	-	.51**	24	60	12	4	.98**
LSD 5%					.25					.22
1%					.36					.32

a. Five spray applications were made at approximately 7-day intervals beginning July 18, 1956. Leaf samples were collected 2 weeks after the fifth spray. Variety Calabrese used.

b. Data are based on 3 samples of 50 leaves collected from upper parts of the plants in each treatment and graded as follows: none, no infection; trace, less than 1/4 of leaf area infected; medium, 1/4 to 1/2 of the leaf area infected; severe, over 1/2 of the leaf area infected.

c. Based on rating each leaf as follows: No infection, 0; trace, 1; medium, 2; severe 3.

* Significant at 5% level; ** significant at 1% level.

Table 2. Field studies on control of bacterial spot of lima beans with three streptomycin sulfate (Agristrep) sprays with and without glycerine[a].

Treatment	Leaves showing various degrees of severity[b]									
	12 days after last application					20 days after last application				
	None	Trace	Medium	Severe	Mean Disease Rating[c]	None	Trace	Medium	Severe	Mean Disease Rating[c]
	%	%	%	%		%	%	%	%	
Streptomycin SO_4 200 ppm + 1% glycerine	73	25	2	-	.30**	66	30	4	-	.39**
Streptomycin SO_4 200 ppm	61	33	6	-	.39**	61	36	3	-	.42**
Check	30	49	21	-	.70	29	56	15	-	.86
LSD 5%					.14					.23
1%					.24					.38

a. Based on 3 100-trifoliate samples from each treatment; graded as follows; No infection, 0 lesions; trace, 1-5 lesions; medium, 6-20 lesions; severe, over 20 lesions.

b. Streptomycin sprays (Agristrep) were applied on August 22, August 27 and September 1 (5-day intervals); Variety Henderson baby lima used.

c. Based on rating each leaf as follows: No infection, 0; trace, 1; medium, 2; severe, 3.

** Significant at 1% level.

BACTERIAL LEAF SPOT OF LIMA BEANS

Three applications of 200 ppm streptomycin sulfate, with and without 1% glycerine, were applied at 5-day intervals, on August 22, 27, and September 1, in a field of lima beans where bacterial spot was present. Sprays were applied with a 200-gallon, 3-row power sprayer on 1/2-acre plots consisting of 24 rows. An unsprayed 9-row plot between spray blocks served as a check. Three samples of 100 trifoliate leaves were collected from each treatment on September 12 and 20, 12 days and 20 days, respectively, after the last application and graded according to disease severity. Data are given in Table 2.

It may be concluded that three sprays of 200 ppm streptomycin, applied at 5-day intervals, gavea significant reduction in bacterial leaf spot of lima beans. The addition of glycerine to streptomycin gave no significant improvement in control over that obtained with streptomycin alone. Since no pod data were taken, the effect of the sprays on yield could not be determined. Considering the cost of streptomycin and the amount of reduction in disease, the value of streptomycin under these conditions is questionable.

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ANGULAR LEAF SPOT OF BEAN IN COLOMBIA[1]

Ovidio Barros[2], Canuto Cardona[3], Ricardo Cardeñosa[4], and R. L. Skiles[5]

Summary

Prior to 1953 angular leaf spot (Isariopsis griseola) of bean was of minor importance in Colombia, but during the next 2 years the disease increased in destructiveness until in the latter part of 1955 it caused heavy losses in the dry bean crop, particularly in the Cauca Valley. The variety attacked most severely was Algarrobo, which had been considered to be highly resistant because in 2 years of testing in the Cauca Valley it had remained free of the disease. As this variety was high-yielding as well, it was widely grown following its release for commercial production in 1954. It appears likely that the cultural practices followed by farmers in the Valley played an important part in the development of the 1955 epiphytotic. Many farmers planted as many as four crops a year without rotation, and in the plantings during the dry periods used row or overhead irrigation, thus both shortening the length of time that the fungus had to survive between crops and supplying the moisture necessary for infection. Since 1955 the years have been dry and angular leaf spot has been of negligible importance in the Cauca Valley.

Angular leaf spot (Isariopsis griseola Sacc.) of bean (Phaseolus vulgaris L.) is known in practically all parts of the world where the crop is grown (1, 2, 3, 5, 11, 12). Although it is usually considered to be a disease of minor importance, in isolated cases it has caused relatively heavy losses (4, 7, 8, 9). In the latter months of 1955 in Colombia the dry bean crop was severely damaged by angular leaf spot, especially in the Cauca Valley, which is the principal bean-producing area of the country. As a result, several previously good varieties can no longer be safely planted, and the cooperative bean-improvement program of the Colombian Ministry of Agriculture and The Rockefeller Foundation has been changed to include the use of some completely new sources of germ plasm with angular-leaf-spot resistance in the breeding program.

The symptoms and the environmental factors that favor the development of angular leaf spot have been described by Cardona and Walker (6). Similar or identical symptoms are observed in Colombia, including the typically angular lesions on the leaves, the large circular ones on the pods, and the stem and petiole lesions (Figs. 1, 2, and 3). The first field symptoms appear on the primary leaves during the earliest stages of growth. However, the disease does not become conspicuous until late flower or early pod set. Defoliation occurs subsequent to the latter stages of growth, and the pods that remain bear poorly developed or entirely shriveled seed.

When the bean improvement program started in Colombia in 1951, angular leaf spot was of very minor importance in the Cauca Valley. Moreover, no information could be found later to indicate that it had ever been important in that region. In experimental plots traces of the disease were occasionally seen in a few varieties during the years 1951-1954, but the principal commercial dry-bean varieties, Panameño and Sangretoro, of the Cauca Valley were disease-free during that period. Several thousand hectares of these two varieties were planted twice each year by the local farmers[6]. The variety Algarrobo, which was new to the

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[5]Associate Plant Pathologist, Colombian Agricultural Program of The Rockefeller Foundation.
[6]The climate in Colombia is such that bush bean can be planted twice a year at elevations below approximately 2000 meters. These growing seasons are termed semesters; the first is called the A semester and the second the B.

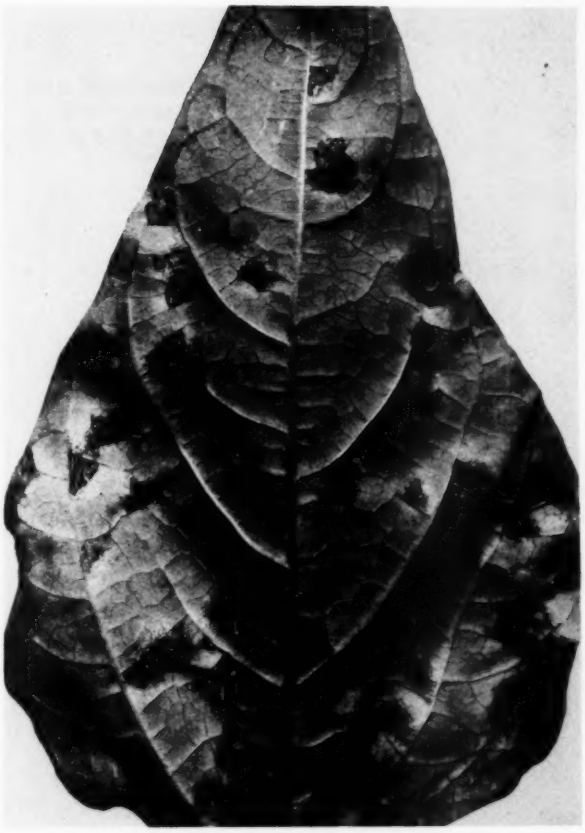

FIGURE 1. The lower surface of a bean leaf showing the typically angular lesions caused by Isariopsis griseola and the formation of coremia in the lesions.

Cauca Valley, was equally free from angular leaf spot through four consecutive growing seasons. In the 1951-1953 yield trials Algarrobo was being observed for potential release to the farmers because it was higher yielding than Panameño and Sangretoro.

In Medellín, which in comparison with the station in the Cauca Valley is higher, cooler, and receives more rainfall, the same three varieties were planted in order to study their reactions to various diseases. At the experiment station near Medellín such foliage diseases as rust (Uromyces phaseoli typica Arth.), anthracnose (Colletotrichum lindemuthianum (Sacc. & Magn.) Scrib.), and bacterial blight (Xanthomonas phaseoli (E. F. Sm.) Dows.) develop rapidly and extensively every growing season, so that good disease readings can be obtained. In 1951 and 1952 angular leaf spot had developed in Medellín only to the extent that "trace" readings were recorded for Sangretoro, Panemeño, and Algarrobo. Through the next 2 years however, the disease increased until the three varieties finally gave consistent readings of "susceptible" or "highly susceptible." During 1953 and 1954 in the Cauca Valley, Panameño and Sangretoro continued to be good commercial varieties with respect to freedom from angular leaf spot, and Algarrobo remained free from the disease in yield trials and increase plots. By the end of the second growing season of 1953 it was decided to increase and distribute Algarrobo to the farmers in the Cauca Valley.

During 1954 and 1955, approximately 45 1/2 metric tons of Algarrobo seed were sold, and the yields of this variety in commercial plantings were consistently higher than those of Panameño and Sangretoro. At the end of the first growing season in 1955 Algarrobo was in such demand that seed harvested by the farmers was being sold to others who wanted to plant it the following semester. Consequently, only a part of the seed was ultimately reaching the markets for consumption.

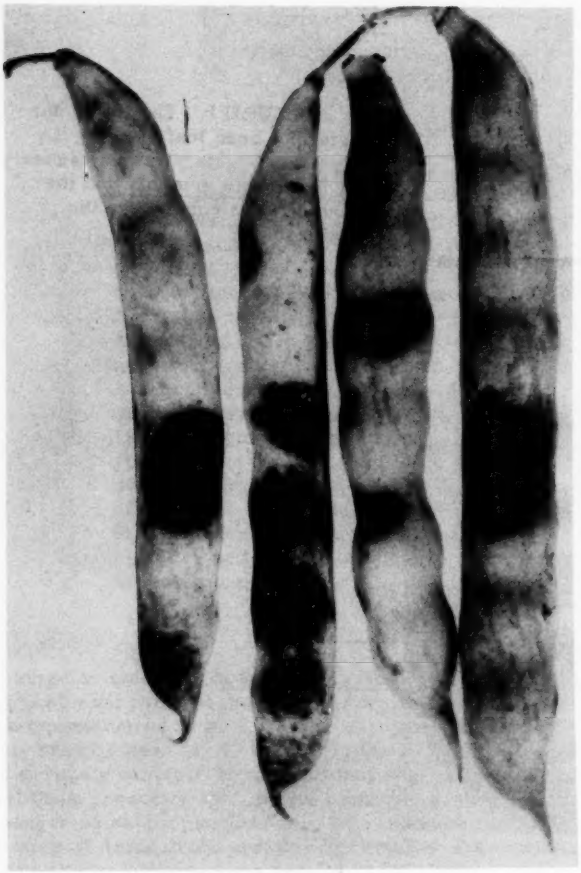

FIGURE 2. Bean pods showing the large, circular, dark lesions caused by Isariopsis griseola.

One farmer prepared his land in the center of the Cauca Valley for furrow irrigation during the dry months of 1955, and also installed a sprinkler-irrigation system. By these means he was able to produce four crops of beans per year, viz. two in the rainy months of March to June and September to December, and two in the dry months of June to September and December to March with irrigation. Algarrobo was planted on this farm in March, July, October, and December of 1955 and on neighboring farms without irrigation in March and October. The March planting was successful, but in the July planting considerable angular leaf spot was observed. There appeared to be no difference between the furrow-irrigated and the sprinkler-irrigated fields with respect to the prevalence and severity of the disease. No Panameño or Sangretoro was planted, so it was not known how these varieties might have reacted. This was the first time that any appreciable amount of angular leaf spot had been observed in the Cauca Valley and the first time also that beans had been grown there with sprinkler or row irrigation in the summer months. It was therefore considered possible that in this region angular leaf spot was a warm-, moderately-dry-weather disease that had never had an opportunity to develop because of previous cultural practices.

It should be noted here, with regard to cultural practices, that there was an increasingly strong tendency on the part of the farmers to plant beans after beans instead of using a better rotation with other crops. This was partly because better returns could be obtained from beans than from other crops, and partly because certain fields were closer than others to the existing sources of water. In the fields in which angular leaf spot was first seen, there had been two previous crops of beans. That this type of rotation played a vital role in the losses

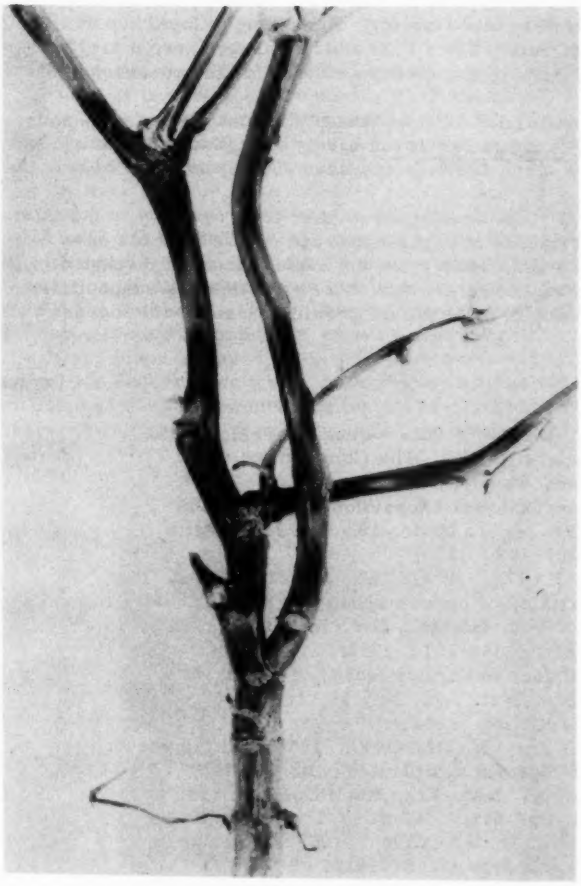

FIGURE 3. A bean plant that has been defoliated by angular leaf spot. Numerous dark lesions caused by Isariopsis griseola can be seen on the stem and petioles.

that were later incurred seems certain, in view of the finding by Cardona and Walker (6) that I. griseola readily survives adverse periods on bean trash left in the field. Furthermore, the short period of time that elapsed between harvest and planting also contributed to the build-up of a large quantity of inoculum.

Earlier, Muller (10) reported from Brazil that angular leaf spot was severe only during protracted dry spells. A similar situation appeared to prevail in the Cauca Valley. In Medellín, however, the disease developed only during the rainy, cooler months, and one or two small plantings with irrigation at that station had failed to demonstrate that the disease could develop to any degree during the warm, dry months. Therefore, the critical conditions for the epiphytotic that was to do so much damage in the rainy months following the summer planting in the Cauca Valley appeared to be 1) poor rotation, 2) close cropping, and 3) sufficient humidity from irrigation for infection to take place.

Algarrobo was planted in the Cauca Valley in October of 1955 on an even greater scale because the rainy periods followed immediately. Many farmers also planted Panameño and small quantities of Sangretoro at the same time. Furthermore, as a result of the increased yields obtained from Algarrobo, many farmers were planting beans in fields that had never before been cultivated. It was apparent by the middle of November that angular leaf spot was going to do a great deal of damage to Algarrobo, and losses of 20 percent were estimated in a few fields before harvest. Loss estimates were based on the number of shriveled or incompletely filled pods.

Simultaneously, angular leaf spot was observed in fields of Panameño. Although the estimated losses were not so high as those expected in Algarrobo, enough disease was present to cause losses never before realized in this variety. According to local reports, Panameño had been grown in the Cauca Valley since 1929 and had always been a healthy variety. There were so few fields of Sangretoro that none were studied for the occurrence of angular leaf spot.

The ultimate result of this epiphytotic in the Cauca Valley was that seed of very poor quality reached the markets and many farmers harvested barely enough to meet their costs of production. Yields were reduced as much as 40, sometimes 60, percent and most of the seed was very shriveled

Since the second growing season of 1955 the semesters have been very dry in the Cauca Valley and there has been very little evidence of angular leaf spot. The farmers have continued to plant Algarrobo and Panameño and, aside from the losses incurred through drought, the harvests have been good. It is known, however, that these varieties are susceptible to angular leaf spot and that, with the return to more humid growing seasons, the disease will again cause severe losses.

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COLOMBIAN MINISTRY OF AGRICULTURE, AND COLOMBIAN AGRICULTURAL PROGRAM OF THE ROCKEFELLER FOUNDATION

A SEED-BORNE BACTERIAL COTYLEDON SPOT OF SQUASH

D. M. Mc Lean[1]

The growing of Blue Hubbard squash (Cucurbita maxima) in northwest Washington for fresh market is restricted mostly to Whidbey Island. After harvest, squash are held in storage and shipped to ready markets during the winter. Crops are relatively free from diseases except for storage rots, which often cause severe losses.

In 1954 our attention was called to a disorder on seedlings, apparently initiated through infected seed. Seeds from different sources were planted in the same field in several locations. Infections were consistently restricted to 10 to 15 percent of the seedlings from one lot of seed. It is not known whether the infected seed was purchased from eastern seedsmen or grown locally. Local farmers usually maintain their own seed stocks from selected squash, but in 1954 some seed had been purchased from an eastern source.

On the cotyledons, spots appeared on the lower side as small water-soaked areas and on the opposite side as indefinite yellow areas (Fig. 1). Eventually the spots were rounded or irregular, often demarcated on the upper surface by a yellowish halo. Only an occasional lesion was observed on the first leaves; infections did not spread from infected seedlings. No trace of the disease could be observed during the season on mature plants or on squash fruits. Early growth of infected seedlings was retarded, and thus maturity and harvest were delayed.

FIGURE 1. Symptoms of a bacterial leaf spot on cotyledons of Hubbard squash.

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It was difficult to determine the seed source, but suspicious seed was planted in the greenhouse where less than 1 percent of the seedlings showed definite cotyledonary lesions.

Plantings on potato-dextrose agar produced a rapidly growing, immarginate, pale-yellow bacterium. However, infections could not be reproduced by spraying seedlings with a bacterial suspension or pricking through droplets of the suspension.

Symptoms in general agree with those described for bacterial leaf spot of squash[2] caused by Xanthomonas cucurbitae (Bryan) Dows. This disease has not been reported from Washington.

UNITED STATES DEPARTMENT OF AGRICULTURE, AGRICULTURAL RESEARCH SERVICE, CROPS RESEARCH DIVISION, MOUNT VERNON, WASHINGTON

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STUDIES ON TRANSMISSION OF VERTICILLIUM WILT OF EGGPLANT IN MICHIGAN[1]

Clyde L. Burton and Donald J. deZeeuw[2]

Abstract

A series of laboratory assays and greenhouse and field experiments to determine the importance of seed transmission of Verticillium albo-atrum causing eggplant wilt was made between 1955 and 1957. Laboratory assays and greenhouse plantings of commercial seed lots showed no infection. The disease was induced readily in plants by inoculation and the organism could be isolated from fruits of these plants but only one fruit produced seeds bearing the organism. The infection, in this case, appears to have been via contact of soil with the fruit. The results of all the experiments lead the authors to conclude that seed transmission of Verticillium wilt of eggplant is extremely unlikely in practice. Some cultural practices minimizing the damages of Verticillium wilt in eggplant are discussed.

Verticillium wilt (Verticillium albo-atrum Reinke & Berth.) is a common disease of eggplant in Michigan. Although losses caused by the disease may be serious in individual fields most damage is incurred through steady infections in infested fields. Foliage of affected plants wilts and turns yellow and the leaves gradually drop off. Stunted growth and vascular discoloration are other common symptoms. Plants infected usually die prematurely unless partially revived by optimum growing conditions.

The literature generally states that eggplant seed is an important agency of Verticillium wilt dissemination. The seeming rapid spread of the disease from one eggplant production area to another plus the reported seed transmission has led many growers of eggplant to believe that the seed stocks procured from the commercial seed houses were contaminated with the wilt pathogen, and were thus the main source of new wilt outbreaks in their fields.

Evidence concerning seed transmission of this pathogen has been conflicting and its importance is uncertain for this growing region. Because of this an investigation was undertaken at the Michigan Agricultural Experimental Station in the Spring of 1955 to determine the relative importance of eggplant seed as a disseminator of Verticillium albo-atrum.

LITERATURE REVIEW

The problem of seed infection by Verticillium has been investigated for several crops. Allen (1) stated that the fungus could survive extended periods in the interior of inoculated cotton seeds without loss of seed viability. Taubenhaus (16) isolated it from the interiors of 199 out of 1441 cotton seeds cultured. Rudolph and Harrison (15) reported, after a 5-year study on cultures from seed produced by diseased cotton plants, that dissemination of Verticillium wilt by seed is highly improbable.

Kadow (9) isolated Verticillium from tomato and eggplant seeds. He concluded that the pathogen was internally and externally seed-borne. Rudolph (14), on the contrary, was unable to find that tomato seeds were an important factor in dissemination. Haenseler (6) in 1921, after making tests on seed taken from the fruit of Verticillium-wilted eggplants, stated that the organism does not advance into the seed of fruit borne on badly diseased plants.

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[2]Respectively, graduate student and Associate Professor, Department of Botany and Plant Pathology, Michigan State University.

METHODS AND PRELIMINARY PROCEDURES

Laboratory: Samples of surface-disinfected and non-disinfected eggplant seeds to be tested for external and internal carriage of Verticillium were plated on 2 percent potato-dextrose agar and Czapek's agar in 9-cm Petri dishes, usually ten seeds per plate. The seeds were half submerged in the agar with sterilized forceps in order to assure recovery of organisms in the interior, and the sprouts were cut aseptically and pressed into the agar shortly after germination. All cultures were held at room temperature for approximately 30 days.

Shake cultures were used to test a large number of seeds at one time, and to provide an additional method for recovery of internal organisms. A potato-dextrose broth was used. This was prepared by adding the extract of 80 grams of cooked potato to 800 ml of distilled water in which 5 grams of dextrose were dissolved. This broth was used at the rate of 125 ml in 250-ml flasks or 250 ml in 500-ml flasks. After seeding the flasks were shaken on the mechanical shakers for about 30 days. Recognizable growth of V. albo-atrum appeared in the flasks within 9 days after seeding the medium with a minute quantity of the organism. This technique also favored seed germination. Repeated trials over 2 years with surface-disinfected seed indicated that the method was acceptable for culturing Verticillium and for recovery of other fungi on seeds. One seed, artificially contaminated by dipping into a mycelial suspension of V. albo-atrum and allowed to dry for from 1 to 24 hours, was capable of producing recognizable growth within 2 weeks among 100 surface-sterilized seeds. Seed germination appeared to be normal under these conditions.

Non-disinfected or aseptically excised seeds produced excessive foreign contamination in shake cultures. Acidifying the medium was of little benefit, and only one out of ten flasks seeded with both non-disinfected seeds and a single artificially contaminated one produced recognizable Verticillium. This same low percentage also held later for controlled experiments.

Preliminary investigations showed that Verticillium dried on the surface of a seed could be washed off with water in shake flasks and recovered by culturing the liquid. Serial dilutions of the wash water aided greatly in identification. Excess fluid was decanted after being allowed to stand on the agar for 10 minutes. The organism was recovered from six of 20 samples containing one artificially contaminated seed and 100 healthy, non-disinfected seeds with this method.

Seeds were surface-disinfected by placing them in a 1:9 dilution of commercial sodium hypochlorite for 2 minutes and then rinsing with sterilized distilled water.

Greenhouse: Greenhouse temperatures generally ranged between 65° and 80° F during the course of the experiments, and produced satisfactory eggplant growth. Potting soil was a sandy loam with added peat and a complete fertilizer. The plants grown to maturity in the greenhouse were transplanted into 12- or 14-inch clay pots and given weekly applications of liquid fertilizer (made from a soluble concentrate N23-P21-K17). Necessary precautions to avoid cross-contamination were observed and the plants were sprayed at weekly intervals to prevent infestations of mites and aphids.

Preliminary experiments were conducted in the greenhouse to find a reliable technique for inoculation of the plants. Dipping the roots of seedlings into a diluted suspension of a 20-day-old shake culture, or probing a long-bladed knife into the soil near the main stem of a well established plant and pouring a small quantity of a diluted 20-day shake culture into the damaged area, were found satisfactory. Young plants could be infected readily by pouring the diluted culture material on the soil without injuring the plant roots, but older plants were more resistant.

LABORATORY ASSAY OF COMMERCIAL EGGPLANT SEEDS

In order to determine the percentage of commercial eggplant seeds that are contaminated or infected by the Verticillium wilt pathogen, eggplant seeds were procured from six seed outlets in different parts of the United States. Black Beauty, Florida High Bush, Fort Meyers Market, New Hampshire Hybrid, and New York Spineless varieties were used. The Black Beauty variety was obtained from all six seed houses and the other varieties from only one.

Four hundred seeds were selected at random from each of the seed packets and half of the seeds from each sample were surface-disinfected. The seeds were planted on potato-dextrose agar and observed frequently for fungal growth. No evidence of Verticillium, internally or externally, was found, and 40 to 60 percent of the seed germinated.

Samples of approximately 2,000 seeds from each commercial variety were shaken in 200 ml of sterile distilled water in 500-ml flasks for 24 hours. Serial dilutions of each shake liquid were poured over potato-dextrose agar and cultured. There were four replications of each dilution. All the samples cultured in this manner were badly contaminated with bacteria and fungi, but none was identified as V. albo-atrum.

Four 600-seed lots of Black Beauty eggplant from each of the various seed outlets were placed in 500-ml flasks containing 250 ml of potato-dextrose broth. Two seed lots were surface-disinfected and two were left untreated. The flasks were shaken for 1 month. The non-disinfected seeds were heavily contaminated and dilution plates of three doubtful cases proved negative for Verticillium. Surface-disinfected seed lots were practically free from bacterial and fungal contamination and Verticillium infection was not present.

Most of the remaining commercial seeds, approximately 20,000, were bulked in one 2-liter flask containing 500 ml of water, in an attempt to locate any trace of V. albo-atrum sufficient to produce infection in healthy plants. The flask was agitated for 2 days and the water decanted from the seeds onto the soil around two newly transplanted eggplants 6 to 8 inches high. More water was added to the seeds repeatedly and used for watering long after the seeds had germinated in the flask. The plants were observed for 5 months, but never developed any evidence of Verticillium wilt.

LABORATORY ASSAY OF SEEDS FROM DISEASED EGGPLANTS

Since no commercial seeds tested proved to carry Verticillium, work was undertaken to procure seeds from plants known to be diseased. One hundred and eighty-nine fruits were harvested from mature Black Beauty eggplants, most of which showed severe external wilt symptoms as well as definite vascular browning in the fruit and stem tissue.

Sixty-seven of the fruits, designated "Lot A", were from plants treated in one of the following ways:

Group No. 1 -- Seedling-inoculated in the greenhouse and allowed to set fruit before transplanting to the field. These plants were approximately 10 months old at time of harvest (10 fruits).

No. 2 -- Seedling-inoculated at the time of transplanting to the field (5 fruits).

No. 3 -- Plants inoculated in the field during the early flowering stage (22 fruits).

No. 4 -- Plants inoculated in the field just after fruit set (20 fruits).

No. 5 -- Plants not inoculated and without symptoms of the disease (10 fruits).

Field inoculations were made by cutting the eggplant roots with a long-bladed knife near the main stem and drenching with a dilute 20-day shake culture of Verticillium. All inoculated plants showed wilt symptoms within 6 weeks. Diseased plants were from one-half to three-fourths the size of healthy controls. The plants infected earliest were smallest and had the smallest fruit.

Lot A seeds were removed aseptically by scraping them into covered plates. They were then allowed to dry before being packaged. Four hundred seeds, half of which were surface-disinfected, from each of the five Lot A groups were plated on potato-dextrose agar. Approximately 400 seeds representative of each of the five Lot A groups were placed in 500-ml flasks with 250 ml of sterilized distilled water and agitated for 48 hours. At the conclusion of this period, three dilutions of the liquid from each flask were assayed on potato-dextrose agar. Approximately 350 seeds from each of the five groups in Lot A were surface-disinfected and placed in 500-ml flasks containing 250 ml of potato-dextrose broth. Each group treatment was replicated eight times. The 40 flasks were shaken for 30 days. Two hundred non-disinfected Lot A seeds from each of the five group treatments were placed in 250-ml flasks along with 125 ml of potato-dextrose broth and shaken for 30 days. This last experiment was repeated five times.

The results of these assays on Lot A seed are summarized in Table 1. None of the seeds in Lot A proved to be externally contaminated or internally infected with V. albo-atrum. Germination of Lot A seeds was normal on agar plates and in nutrient shake cultures, except for group No. 1.

Table 1. Assays for the presence of Verticillium albo-atrum in eggplant seed from fruits of infected plants.

	Lot A Seeds From:									
	(1) Plants inoc. in Green-house		(2) Plants inoc. at transplant time		(3) Plants inoc. at flower time		(4) Plants inoc. after fruit set		(5) Plants not inoc.	
Assay	D[a]	ND	D	ND	D	ND	D	ND	D	ND
Plating on PDA	0/200[b]	0/200	0/200	0/200	0/200	0/200	0/200	0/200	0/200	0/200
PD broth shake	0/2800	0/2800	0/2800	0/2800	0/2800	0/2800	0/2800	0/2800	0/2800	0/2800
PDA assay of seed washings		0/400		0/400		0/400		0/400		0/400

Lot B Seeds: 87 infected fruits of 122 infected plants.

	No. fruits (seed assay)	Seeds assayed per fruit	No. fruits with infected seed
Assay of aseptically removed seeds			
(1) PDA plates	122	50	1[c]
(2) Czapek's plates	122	50	1
(3) PD broth shake	122	10	1
Assay of dried, surface disinfected seeds			
(1) PD broth shake	122	10-15	0
(2) Ground seed shake	122	10	0

[a] D = surface disinfected, ND = seed not disinfected.
[b] The numerator represents the number of seeds infected and the denominator the number tested in each case.
[c] Fruit and seed lot No. 3V3, infection of seed ranging between 40 and 70 percent in this assay and other trials.

One hundred and twenty-two fruits designated "Lot B" were harvested from plots known to have been contaminated by the wilt organism. Each fruit was recorded and its condition noted at time of harvest. The surface of each fruit was washed in running water for several minutes to remove field soil, and the portion just above the calyx was removed aseptically. The vascular system was inspected for browning and several bits of the brown vascular tissue from each fruit were plated on potato-dextrose agar and Czapek's agar. Eighty-seven of the 122 fruits indexed in this way contained Verticillium of the pathogenic type. Cultures agreed with published descriptions of V. albo-atrum, microscopically (4, 7, 13) and in pathogenicity when used to inoculate clean seedlings. Reisolation from infected seedlings gave the original type of the organism.

One hundred seeds from each fruit of Lot B were excised aseptically and individually. Fifty of these were plated on potato-dextrose agar and 50 on Czapek's agar, ten seeds per plate. Transfer forceps were sterilized between successive seed transfers. Another group of ten seeds from each fruit was placed in 250-ml flasks containing 125 ml of potato-dextrose broth and shaken for 1 month.

All of the seed assayed in these trials, with one exception, were negative for Verticillium. Seeds from one fruit, hereafter designated 3V3, showed characteristic colonies of V. albo-atrum on from 40 to 70 percent of seeds plated on either of the solid media. The organism was also recovered readily from the liquid shake culture. The pathogen was identified microscopically, and by inoculation of healthy eggplant to produce the disease. The same organism was recovered from the inoculated plants. The remaining seeds of each separate fruit were removed, dried and stored.

Germination of Lot B seeds plated on agar was considered too low to give an accurate indication for internal disease carriage of the wilt organism, if emergence of the sprout were to aid in release of the fungus. Ten to 15 seeds from each fruit were surface-disinfected, placed in flasks containing potato-dextrose broth, and shaken for 30 days. Another ten seeds from each fruit were surface-disinfected, dried, and ground coarsely to permit good exposure of the interior of each seed. The ground seeds of four fruits were mixed with sterilized water in each flask, shaken for 2 hours and then poured over potato-dextrose agar in Petri dishes. Excess liquid was removed after 15 minutes.

Lot B seed that had been surface-disinfected and shaken in potato-dextrose broth for 30 days germinated but the culture medium remained free from the pathogen. No Verticillium was cultured from the shake liquor of ground seeds. The results of these assays on Lot B seed are summarized in Table 1.

Two additional experiments were made with the contaminated (3V3) seeds to determine if the organism were seed-borne internally. Sixty 3V3 seeds were surface-disinfected, and three groups of ten seeds were shaken for 60 days in potato-dextrose broth. The remaining 30 seeds were placed in Petri dishes on moistened blotters and allowed to germinate. After the seeds had germinated (six seeds), cooled liquid potato-dextrose agar was poured over the sprouts. No Verticillium developed in the shake flasks or in the sprout plates.

The unique behavior of 3V3 seeds prompted a comparison of the harvest notes on fruits of Lot B. This fruit (3V3) was above average in size, well matured, of good color, and contained an abundance of seeds. On examination the seeds, however, were seen to be only fair in quality (color, size, and plumpness). The vascular system of the fruit was filled with the wilt organism, as shown by the isolations from fruits previously described. A rather small crack was noted at the seed end, which appeared to have been caused by a mechanical injury of some sort. Decomposition had begun in this area and it was infested with small insects. The 3V3 seed that was harvested for experimental use was taken from non-decomposing pulp and the decayed section was discarded. The seed from this fruit germinated poorly.

FIELD EXPERIMENT WITH SEEDS FROM DISEASED PLANTS

This experiment was designed to study possible transmission through lightly infected seed under natural conditions. Since the laboratory seed assays failed to disclose any Verticillium in the seed, the field experiment also acted as a partial check on laboratory techniques. Seedlings from Lot A seeds from diseased eggplants were transplanted into four noncontiguous plots on the Plant Pathology farm. The plots were selected because they had no known history of Verticillium wilt.

Nine plants from each of the four Lot A seed groups No.'s 1 to 4 inclusive, were represented in each of the four plots. Control plants grown from healthy seeds were included in each plot. The plants were allowed to grow to maturity and produce fruit, with normal field

care. After harvest all plants were cut at the base of the stem and the vascular systems examined for signs of the disease.

Two of the four plots proved to have been previously contaminated with Verticillium, as indicated by the high incidence of wilt in control plants. Two of the plots produced plants without external or internal wilt symptoms. Soil samples from the root areas of each of the plots were brought into the greenhouse for test. Three healthy eggplant seedlings were planted in a composite mixture of the infested soils and three in a composite mixture of the apparently clean soils. No disease developed in the clean soil series but all the plants in the infested soils were displaying wilt symptoms within 3 months. These results indicated that eggplant is very susceptible to attack by the wilt organism if it is naturally present in the soil, but it also showed that seed taken from fruit of diseased plants can give rise to apparently healthy eggplants.

GREENHOUSE ASSAY

Greenhouse experiments were made to investigate the importance of planting naturally Verticillium-contaminated seeds in various soils, and to determine whether apparently non-contaminated seeds taken from diseased plants and diseased fruits were of any importance in disease transmission.

Five plants from each Lot A seed group, No.'s 1 to 5, were grown individually in pots of sterilized soil in the greenhouse for 3 months. At the end of this time all the plants were cut down and the vascular systems were examined for signs of the pathogen. There was no visual evidence of the pathogen, nor could the fungus be recovered from any plants by isolation on potato-dextrose agar.

Various blends of seeds from Lot B (diseased fruit) and seeds from non-diseased plants were planted in 14-inch pots in the greenhouse to ascertain if contaminated (3V3) seeds, and seeds from diseased fruits (assayed as non-contaminated) transmit the disease to the growing plant. Fifty or 100 seeds were planted in each pot. Steamed soil and non-steamed soil, previously indexed and found free from the wilt fungus, were used. The different soils were employed to determine whether activity of small quantities of V. albo-atrum, such as a seed could carry, was hindered more by a new microbial population following sterilization than by the native microflora of the field.

Contaminated (3V3) seeds, non-contaminated seeds from diseased fruit, or seeds artificially contaminated by dipping in a weak suspension of the wilt organism and drying, were used in transmission studies in various proportions with surface-disinfected clean seeds. Clean seeds were used for indexing soils for contamination.

The seed combinations were mixed before planting to randomize the initial loci of infection. Eight pots of contaminated soil planted with clean seeds were included as controls.

When seedlings were approximately 2 to 3 inches in height, they were thinned by trimming the tops of each plant just below the lowest leaf with a sterilized razor blade. The remaining stem and root system was left as a potential inoculum source. Successive thinnings were made in the same manner as plants grew larger, until one plant remained in each pot. At the end of the third month roots were damaged by cutting to increase disease susceptibility. The experiment was terminated at the end of 5 months. Figure 1 illustrates some of the symptoms of Verticillium wilt obtained on larger eggplants in the greenhouse in the course of the experiment.

The above methods were employed because preliminary experiments and the literature indicated that root anastomosing (8), decaying infected roots (8, 11, 12), and time of exposure were significant factors in induction of the disease. Maximum opportunity was given for latent seed infections to develop, spread in the soil, and infect other plants.

At the end of 5 months many plants had produced from one to several fruits. Many of the smaller plants in certain pots were dying or already dead from the disease. The remaining single plants in each pot at the end of the experiment were cut down, and the vascular system examined for indications of the disease. The results of the experiment are summarized in Table 2.

Seeds from the contaminated (3V3) seed sample or from non-contaminated seeds of diseased fruit did not induce disease, either in steamed or non-steamed soil. Artificially contaminated seeds, on the other hand, were capable of transmitting the disease under similar circumstances. The inoculum load appeared to be of some importance, since all ten pots planted with ten artificially contaminated seeds had contained diseased survivors while seven of ten were diseased when one artificially contaminated seed was used with 99 clean seeds.

FIGURE 1. Symptoms of Verticillium wilt of eggplant in the greenhouse. (A) Early symptoms of wilt. Marginal wilt of leaves without chlorosis in a plant infected late. (B) Chlorosis and partial recovery from flagging in a plant infected early. (C) Severe flagging following early infection. Plants in this condition usually are unable to recover enough to produce a crop. (D) Death following extreme chlorosis and flagging. Plant infected early.

Table 2. Verticillium transmission trials using contaminated eggplant seed in steamed and non-steamed soil.

Soil used	Replicates (14 inch pots)	Find and number of various seeds used in each pot						Seeds per pot	Diseased plants
		(3V3) Contaminated		Non-contaminated seed of diseased fruit		Seed artificially contaminated	Clean seed		
		D[a]	ND	D	ND		D		
Steamed	2	--	100	---	---	--	---	100	0/2[b]
	2	50	--	---	---	--	---	50	0/2
	5	--	10	---	---	--	90	100	0/5
	5	10	--	---	---	--	90	100	0/5
	5	--	--	---	100	--	---	100	0/5
	5	--	--	100	---	--	---	100	0/5
	5	--	--	---	---	10	90	100	5/5
	5	--	--	---	---	1	99	100	3/5
	3	--	--	---	---	--	100	100	0/3
Not steamed (clean)	2	--	50	---	---	--	---	50	0/2
	4	--	10	---	---	--	90	100	0/4
	5	--	--	---	100	--	---	100	0/5
	3	--	--	100	---	--	---	100	0/3
	5	--	--	---	---	10	90	100	5/5
	5	--	--	---	---	1	99	100	4/5
	3	--	--	---	---	--	100	100	0/3

[a]D = surface disinfected, ND = not disinfected.
[b]Numerator = number Verticillium diseased plants, denominator = number of plants remaining at five months (1 per pot after thinning).

The eight controls using infested soil all had diseased survivors.

In order to test for traces of Verticillium that may have left the seed without causing infection in the surviving plant, all of the pots that were previously negative and seven of the pots in which Verticillium was transmitted were replanted. One hundred clean, untreated commercial seeds were planted in each pot without disturbing the previous roots and stumps. Plants were thinned periodically during the following 5 months, as described previously, to leave one per pot at maturity. Each remaining plant was cut and examined for disease. Evidence of Verticillium wilt was only found in the seven pots of infested soil that had previously produced diseased plants.

DISCUSSION

Although eggplant seeds had been assumed to be a carrier of the Verticillium wilt fungus following the work of Kadow (9), and isolations from infected seeds had been reported (16) in other crops, it was uncertain whether seed contamination was of importance in establishment of the disease in Michigan. The disease is locally important in many places and commercial seed has been suspect. Because of the possibility that seed transmission could cause contamination of new fields or increase disease in partially contaminated fields many attempts were made to recover V. albo-atrum from eggplant seeds.

Commercial seeds appear likely to be clean since none of the assays employed in laboratory or greenhouse revealed the presence of the fungus. The only possibility of contamination in commercial seeds would seem to be through fruit pulp contamination and careless seed harvest.

Nearly all attempts to induce infection of seeds with the wilt organism by growing fruits on diseased plants were failures. Infection of plants was easily achieved and a high percentage of fruit on these plants contained the fungus within the vascular tissue, but only one of the infected fruits produced seeds that carried Verticillium. This fruit was cracked, and the aggregation of Verticillium hyphae in the vicinity of the crack together with partial decomposition of the fruit, makes contamination from the soil a distinct possibility. Had infection taken place via the vascular system it is difficult to explain why none of the other infected fruits produced infected seeds.

The presence of the organism on seeds, however, did not insure its transmission under natural conditions. The greenhouse indexing using these seeds showed that they did not produce infection in the seedlings or in plants near by. Every opportunity was given for latent or minimal infection to develop. Seed infestation, in this case, must have been external or in a vulnerable condition to be so ineffective in inducing disease. Whether the soil was steamed or not also appeared to be immaterial, and it is thus concluded that competing organisms were not a factor in our soil.

Very small amounts of the fungus tissue were shown to be capable of producing identifiable growth in numerous tests in the laboratory. Artificially inoculated seeds transmitted the disease in the greenhouse when planted in as low a proportion as 1 to 99 clean seeds in either steamed or non-steamed soil. This is hardly evidence for seed transmission in nature, since the inoculum was external and easily killed by fungicides. Natural infection of seeds would almost certainly need to be via the vascular system if sound seed harvesting practices were followed.

The results of numerous laboratory, greenhouse, and field experiments over a 3-year period requires the conclusion that seed transmission of Verticillium wilt in eggplant is highly improbable.

Verticillium is adapted to a wide range of plants, including trees, flowers, vegetables, bushes, field crops, and weeds, and the pathogen is able to survive 14 or more years in the soil (17). Once the organism has been introduced into an agricultural area, all susceptible crops will sooner or later be subjected to infection. During the course of this work certain cultural practices were found to aid in producing a satisfactory eggplant crop in the presence of the organism. For the most part these practices conform to horticultural recommendations for eggplant (3). Where the disease is known to be present in the neighborhood, good culture may alleviate the worst effects of the disease and make it possible to grow a profitable crop.

Vigorous seedlings that have been grown in clean soil or sterile soil should be used for transplanting. Since the eggplant is favored by warm soil and the pathogen favored by cool soil (2, 5, 10), it is advisable to delay transplanting until the soil is warm. In Michigan this will usually be after June 15. Fields without a history of Verticillium wilt should be chosen

where possible (5, 17). Eggplants should not be planted following eggplants, tomatoes, potatoes, peppers, strawberries, or brambles. Low areas of fields or other locations into which the wilt organism may have been introduced by drainage are generally to be avoided. Any practice that will minimize root injury at transplant time is also to be recommended. A high nitrogen level should be maintained in the soil by the use of a starter solution at transplanting time and by side dressing with nitrogen after the first fruits are set. It is desirable to protect the transplants from sun and wind for several days and to supply adequate water for the first several weeks. Only shallow cultivation to control weeds should be used, because root injuries promote serious infection.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, MICHIGAN STATE UNIVERSITY

CURRENT SEASON AND RESIDUAL EFFECTS OF VAPAM SOIL TREATMENTS FOR CONTROL OF VERTICILLIUM WILT OF POTATOES[1]

Roy A. Young and W. J. Tolmsoff

Summary

In trials conducted over a 3-year period Vapam at rates of 160 or more pounds per acre was effective in controlling Verticillium wilt of potatoes. When Vapam was blade-injected into the soil 6 inches deep at rates of 160, 165, or 190 pounds per acre increases of more than 100 sacks (5 tons) per acre resulted. Lower rates of application, from 40 to 120 pounds, resulted in significant yield increases but were far less effective.

Residual or carryover effects were demonstrated when potatoes were replanted in test plots that had been treated and planted to potatoes in the previous year. Yields from soil treated with 190 or 165 pounds of Vapam per acre were approximately 50 sacks per acre greater than yields from the untreated check plots.

INTRODUCTION

Verticillium wilt, often referred to as the early maturity disease of potatoes, causes serious reductions in potato yields in many parts of the country. An aerial survey of potato fields in the Klamath Basin in Oregon indicates that the yield from 10,000 acres of potato land was reduced by at least 500,000 sacks in 1957. This estimate is based on a comparison of condition of the fields when surveyed on September 5 with condition of experimental plots, and on subsequent yield data from experimental plots. Control of Verticillium wilt by a pre-planting soil treatment with 190 pounds of Vapam per acre was reported in 1956 (4). Subsequently trials have been conducted to determine the effectiveness of different levels of Vapam and the duration of the beneficial effects.

METHODS

General: An area of Verticillium-infested sandy loam soil near Klamath Falls, Oregon was used for all of the following trials unless otherwise indicated. Prior to application of chemicals the soil was plowed, disced and packed. Vapam (sodium N-methyl dithiocarbamate dihydrate) was injected into the soil approximately 6 inches deep with a blade injector designed and built by Jensen and Page (2), and the soil was packed with a roller culti-packer immediately after treatment. Mylone (3, 5-dimethyltetrahydro-1, 3, 5, 2H-thiadiazine-2-thione) was applied to the surface of the soil with a fertilizer spreader, then double-disced into the soil. Plots were treated early in May and planted with Russet Burbank potatoes approximately 10 days later. Temperature of the soil at a depth of 6 inches was 58°F in 1956 and 61°F in 1957. Vapam was furnished by Stauffer Chemical Company and Mylone by Union Carbide Chemicals Company. Yield data were analyzed using the multiple range test of Duncan (1).

Dosage Rate Trials: In 1956 Vapam was tested at rates of 40, 53, and 165 pounds per acre and Mylone was tested at the rate of 200 pounds per acre. Each treatment was applied to three replicates 50 by 100 feet. In 1957, rates of 40, 80, 120, and 160 pounds of Vapam per acre were tested. Each treatment rate was applied to three replicates 48 by 600 feet. Yield samples were taken in 1956 from the inner 30 feet of rows in each 50-foot replication. In 1957 three samples were taken from 30-foot sections of row at intervals of 100 feet in each replication.

Residual Effects: In 1956 and 1957, plots originally treated and planted to potatoes in 1955 and 1956, respectively, were again planted to potatoes to check for residual effect of treatments made in the previous year.

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[1]Technical Paper No. 1110, Oregon Agricultural Experiment Station.

RESULTS

Dosage Rate Trials

At all rates tested in 1956 and 1957 there was a decrease in the severity of visual symptoms of the disease with the most striking effects apparent in plots treated with 160 or more pounds of Vapam per acre. A significant increase in yield resulted from the 40-pound treatment (Tables 1 and 2). Results from the higher rates of treatment were far superior however, with increases of 100.5 and 120 sacks per acre occurring in 1956 (Table 1) and 1957 (Table 2). Mylone was much less effective than Vapam.

Table 1. Relative effectiveness of chemical soil treatments for control of Verticillium wilt of potatoes as indicated by yield in 1956.

Treatment	Average Yield[a] cwt/acre	Significance[b] 5%	Increase Over Check
Vapam 165 lbs/a	344.4		100.5
Vapam 53 lbs/a	297.2		53.3
Mylone 200 lbs/a	285.4		41.5
Vapam 40 lbs/a	284.1		40.2
Untreated Check	243.9		

[a]Average of 3 replications, 5 samples per replication.

[b]Any two means not bracketed by the same line are significantly different.

Table 2. Relative effectiveness of different rates of Vapam in controlling Verticillium wilt of potatoes in 1957.

Treatment pounds Vapam/acre	Total Yield[a] cwt/acre	Significance[b] 5%	Yield[c] cwt. No. 1/acre
160	319.0		186.8
120	242.0		151.0
80	260.0		145.7
40	234.0		132.1
Untreated Check	199.0		99.7

[a]Average of 3 replications, six samples per replication.

[b]Any two means not bracketed by the same line are significantly different.

[c]Graded after 6-weeks in storage.

Vapam was tested also on a Verticillium-infested muck soil that was subsequently planted to White Rose and Russet Burbank potatoes. Vapam was applied at rates of 50, 100 and 200 pounds per acre on April 12 and potatoes were planted on May 25. Temperature of the soil on the day of application was 46° F at a depth of 6 inches. Symptoms of Verticillium wilt developed in August. The treatments had no observable effects on disease development or yield. Lack of effectiveness may have been due to the low soil temperature at the time of treatment or to the ineffectiveness of Vapam on muck soil at the treatment rates employed.

Residual Effects

Vapam alone and in combination with Terraclor (85% wettable pentachloronitrobenzene, supplied by Olin Mathieson Chemical Corporation) was tested in 1955 for effectiveness in controlling Verticillium wilt of potatoes (4). The combination treatment was employed to test the assumption that Vapam would act quickly in reducing the population of a pathogen and that the more stable Terraclor might suppress reestablishment of the pathogen over a period of several months. Differences in yield of potatoes from plots treated with Vapam alone and in combination with Terraclor were not significant. Yields from the Vapam-treated plots were approximately four times the average yield from untreated plots. The 1955 plot area was replanted to potatoes in 1956 to test for residual effects of the treatments.

The onset of symptoms was delayed in 1956 in plants grown on soil treated with Vapam in 1955, but differences in foliar symptoms between plants on treated and untreated soil were not as striking as in 1955. When yield data were recorded however, significant increases in yields were noted from all of the treated plots (Table 3).

Table 3. Residual effects of soil treatments for control of Verticillium wilt of potatoes as indicated by 1956 yields from plots treated in 1955.

Treatment	Yield[a] cwt/acre	Significance[b] 5%	Increase Over Check cwt/acre
Vapam 190 lbs/a PCNB 100 lbs/a	264.7		85.6
Vapam 190 lbs/a	228.9	|	49.6
Terraclor 100 lbs/a	208.1	| |	29.0
Untreated Check	179.1	|	

[a]Average of 3 replications, 5 samples per replication.

[b]Any two means not bracketed by the same line are significantly different.

The yield in 1956 from plants grown on soil treated with Vapam in 1955 was approximately 50 sacks per acre more than from plants grown on untreated soil. The highest yields were recorded from plots that had received a combination treatment of Vapam and Terraclor in 1955.

In 1957 slight differences were apparent in development of foliar symptoms in potatoes growing on soil treated in 1956 with 165 pounds of Vapam per acre as compared with those on untreated soil. There was a significant increase in yield also from potatoes on plots treated with 165 pounds per acre of Vapam in 1956 as compared with potatoes grown on untreated soil.

DISCUSSION

In trials conducted over a 3-year period, Vapam was effective in controlling Verticillium wilt of potatoes, when applied to the soil at rates of 160, 165, and 190 pounds per acre. Lower rates of application, from 40 to 120 pounds per acre, usually resulted in significant increases in yield, but were far less effective than rates of 160 pounds or more. Under the conditions of these experiments use of 160 pounds or more of Vapam per acre seems necessary to obtain maximum benefits. Rates of application higher than those employed to date

might result in even greater yield increases since even plants growing on soil treated with 160 pounds per acre became yellow and died prior to frost as a result of Verticillium infection.

Residual or carryover effects were demonstrated when potatoes were planted in test plots used in the preceding year. Yield increases of approximately 50 sacks per acre were recorded from potatoes grown on soil previously treated with 190 or 165 pounds of Vapam per acre. These effects probably can be attributed to reduction in Verticillium inoculum potential by Vapam treatment (3) since by the end of the season Verticillium could be cultured from stems of all plants in the plots regardless of symptom development. The residual benefits occurred even though the microsclerotia-infested potato plants from the previous year's crop were disced into the soil.

Residual effects have considerable bearing on the development of an economically feasible treatment program. Even yield increases of 100 sacks per acre are not sufficient to pay the cost of treatments of 160 pounds of Vapam per acre. If a treatment can restore soil to good agricultural usage for a period of years the cost of such a treatment can be amortized over a period of several years rather than be charged against one year's crop production. Trials are currently underway to determine 1) the effectiveness of 2 successive years treatment of soil with different rates of Vapam, 2) residual effects from such treatments and, 3) the effectiveness of various crop residues in suppressing the build-up of inoculum in the soil.

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SILVER SCURF ON MUCK POTATOES

L. V. Busch[1]

The last few years have seen a tremendous increase in the number of washed potatoes offered for sale in polyethylene bags. This is particularly true of potatoes grown on organic or muck soils. Washing, however uncovers diseases and imperfections which previously had not been noticed. The sales appeal of this product depends on the production of well-shaped, clean tubers free from objectionable blemishes. Accordingly, silver scurf, which occurs as unsightly circular to irregular silver brown to dark brown lesions on the surface of the tubers, has suddenly become a major problem.

Owing to the concern expressed by the growers and packers of table potatoes from Bradford Marsh, Ontario, and the widespread prevalence of silver scurf on registered seed potatoes, a study of the causal organism, Helminthosporium atrovirens (Harz.) Mason & Hughes (Spondylocladium atrovirens Harz.), and of the control of the disease was undertaken in the spring of 1957.

Tuber treatment for the control of silver scurf has been tried by several workers without much success. In most cases severe damage was caused to the eyes of the potatoes before adequate disinfection was accomplished. It was felt therefore that an evaluation of some of the newer fungicides now available would be worth while.

The method of assessing the effectiveness of the chemicals used was as follows: Discs were cut from lesions on the surface of infected potatoes which had been treated with the test chemical. A large cork borer (#10) was found to be admirably suitable for this purpose. These discs, which were 1/4 inch thick, were placed on damp quartz sand in Petri plates, five treated pieces around the edge with a control disc in the centre of each dish. The dishes were left at room temperature for 14 days, then they were examined with a binocular microscope. The amount of sporulation present was then rated as abundant, moderate, sparse, or nil.

The concentrations of the test solutions in most cases followed the manufacturer's directions printed on the container. Ten infected tubers of the Katahdin variety were washed and placed in 2-gallon crocks containing a solution of the test chemical. It was found that 1 litre of liquid would just cover ten medium sized tubers. One disc was removed from each of the ten tubers, as described above, and the ten discs so obtained were distributed between two plates. Tubers from which the discs were taken were kept in storage until the extent of the damage caused by the chemical to the sprouts could be noted. This damage was assessed as severe, slight, or none.

As with Burke (1), the absence of sporulation was accepted as a criterion of disinfection. This was considered valid since the discs from any treatment rated as nil at 14 days and kept for several weeks longer never showed any signs of sporulation. All cases with a rating of nil or sparse were repeated at later date.

RESULTS

An examination of Table 1 reveals that of the 21 chemicals used only five, namely Puraseed[2], Semesan Bel[3], Karathane[4], Terraclor[5], and Manzate[6], fulfilled our requirement for a successful disinfectant, that is, one that would prevent sporulation with little or no damage to the eyes of the potatoes. All five of these chemicals were retested with similar results. It is interesting to note that not only did the treated discs in the Terraclor plate fail to sporulate, but the control disc also was affected by the presence of the chemical; possibly a volatile fungistatic substance was given off from the treated discs which acted at a distance to prevent sporulation on the control.

Whether the use of seed-piece treatment will be of any value in the control of the disease remains to be seen. Burke (1) and Schultz (2) are inclined to the view that most of the in-

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[1]Plant Pathologist, Department of Botany, Ontario Agricultural College, Guelph, Ontario, Canada.

[2]Phenyl amino cadmium dilactate; phenyl mercury formamide.

[3]Hydroxymercurinitrophenol; hydroxymercurichlorophenol.

[4]Dinithophenylcrotonate.

[5]Pentachloronitrobenzene.

[6]Manganese ethylene bis dithiocarbamate.

Table 1. Evaluation of certain chemicals for effect on sporulation of Helminthosporium atrovirens, and for phytotoxicity rating.

Chemical	Amount Per Litre of Water	Length of Soak in Hours	Sporulation on 10 Discs					Damage to Eyes
			Time After Treatment	Rating[a]				
				A	M	S	N	
Gallotox	10 ml.	18	14	-	-	-	10	Severe
Puraseed	1 gm.	18	14	-	-	-	10	Slight
Oxyquinoline Benzoate	5 gm.	18	14	8	2	-	-	Severe
Vancide	10 ml.	18	14	8	2	-	-	None
Thioneb 50W	5 gm.	18	14	3	2	5		None
Orthocide 50	2.4 gm.	18	10	10	-	-	-	None
Agrostrep	.1 gm.	18	10	10	-	-	-	None
Panogen	10 ml.	18	10	-	-	-	10	Severe
Mecuric Chloride	1 ml.	18	10	-	-	-	10	Severe
Javex	143 ml.	18	10	-	-	10	-	Severe
Formalin	10 ml.	17	10	-	-	10	-	Severe
Formalin	20 ml.	17	10	-	2	8	-	Severe
Acti-dione	1.25 gm.	17	10	-	8	2	-	Slight
Dithane	2 gm.	17	11	3	7	-	-	None
Fermate	2 gm.	17	11	-	1	9	-	Slight
Zerlate	1.25 gm.	17	11	6	4	-	-	None
Terraclor	20 gm.	17	11	-	-	-	10	None
Manzate	2.5 gm.	17	11	-	-	-	10	None
Spergon	6 gm.	1 min.	10	8	-	2	-	None
Semesan Bel	16 gm.	1 min.	10	-	-	1	9	None

[a] A = abundant
M = moderate
S = slight
N = none

fection comes from inoculum carried by the seed piece. If this is true then it should be possible to control silver scurf of potatoes commercially by the judicial use of tuber treatment. However soil inoculum, as shown by Taubenhaus (3), undoubtedly plays a large part in the epidemiology of silver scurf. Providing that sporulation is a good criterion of disinfection, then field results obtained from treating seed potatoes with Semesan Bel, Karathane, Puraseed, or Terraclor this past growing season not only supports Taubenhaus but also raises doubts as to the value of such treatments for the control of silver scurf.

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DEPARTMENT OF BOTANY, ONTARIO AGRICULTURAL COLLEGE, GUELPH, ONTARIO, CANADA

THE EFFECT OF PENTACHLORONITROBENZENE (PCNB) ON MUSHROOM PRODUCTION[1]

R. N. Goodman[2]

Summary

The fungicide PCNB at 250 ppm or higher applied 24 hours after casing soil has been put on delays production and limits yield of mushrooms. If the material is applied after harvest of the first break concentrations as high as 1000 ppm do not affect yield adversely.

The efficacy of pentachloronitrobenzene (PCNB) in eradicating established infections of mushroom mildew, Dactylium dendroides, has recently been described (1, 2).

There have been, since then, however, a number of reports from mushroom producers that PCNB has a severe inhibitory affect upon mushroom development. These effects are described as a delay in production and a reduction in yield, with some producers reporting virtual sterilization of the treated beds.

The author has had an opportunity to survey a few cases of PCNB injury of the type described above. In most instances the beds that were treated with the fungicide had demonstrated a good-average mycelial activity in the compost. Further, it was noted, without exception, that where PCNB had delayed and/or reduced production, the material had been applied soon after "casing" or at least before any mushrooms had been produced.

Turning once again to reports of Stiller and co-workers (2) and Goodman (1) it is to be noted that applications of PCNB at 1000 ppm had not been phytotoxic. In these instances however, the fungicide was applied after the first break or to beds that had been in production and had been "recased".

It was apparent, nevertheless, that additional information regarding the nature of PCNB injury and methods for the safe application of this chemical were needed.

METHODS

The fungicide PCNB was applied at concentrations of 250, 500, and 1000 ppm to beds 24 hours after the 1 1/2-inch layer of casing soil had been put on. Plots of 30 square feet were utilized, wherein one half was treated and the remaining half maintained as a control. Three plots were established for each concentration.

In the second experiment, one half of a small house of approximately 2000 square feet of bed space was treated with PCNB; the remaining half was left untreated as a control. The PCNB was applied at 1000 ppm 2 weeks after the beds had been in production and the first break had been harvested. The plot which received the PCNB had at that time produced 7 percent more mushrooms than the plot which was to be left as a control. At the time of application an examination of apparent vigor and extent of spawn development showed both halves to be comparable.

In both experiments PCNB was applied under low pressure through conventional watering apparatus, and an attempt was made to wet the beds to a depth of approximately 1/2 inch.

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[1] Journal Series Paper No. 1846. Approved by the Director of the Missouri Agricultural Experiment Station

[2] Associate Professor of Horticulture, University of Missouri, Columbia, Missouri.

RESULTS

Results from the first experiment were most conclusive in that all three concentrations both delayed and severely reduced production. Mushrooms that eventually grew were abnormally large. This delay persisted for more than 3 weeks, after which a few large mushrooms, developing at the compost-casing soil interface, appeared. Figure 1 presents the effect of PCNB at 250 ppm applied 24 hours after the casing soil had been put on.

Table 1 presents the weekly cumulative production of the treated and control plots of the second experiment. These data indicate that PCNB applied at 1000 ppm after the first break had been harvested did not reduce yield.

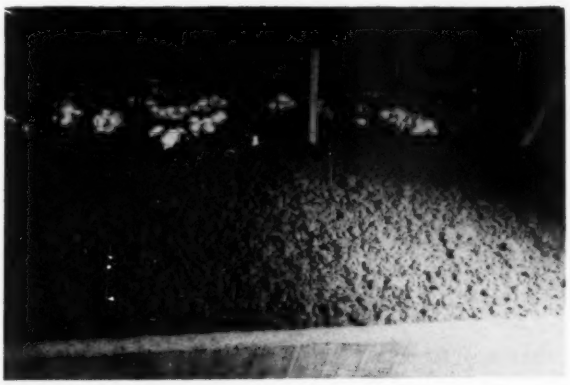

FIGURE 1. Effect of 250 ppm. PCNB on mushroom production. Treated half in foreground, control in background.

Table 1. Effect of PCNB on mushroom production.

Treatment	Weekly cumulative production											Production pounds per square foot
	1	2*	3	4	5	6	7	8	9	10	11	
PCNB 1000 ppm	219	825	1391	1644	1893	2115	2160	2343	2451	2493	2565	2.51
Control	462	762	1221	1560	1833	1929	2142	2244	2361	2469	2481	2.43

*PCNB applied at end of second week of production -- calculated termination of first break.

DISCUSSION

The experiments described herein establish the fact that PCNB, applied at 250 ppm or higher to mushroom beds 24 hours after casing, delays and curtails production. The mechanism involved is as yet unknown. However, Stoller (2) has reported that as little as 50 ppm PCNB admixed with casing soil delayed production and reduced yields. Characteristic sub-casing soil development of mushrooms in beds treated with PCNB prior to harvest also suggests that the chemical inhibits development of the fungus in the casing soil. An apparent paradox exists however, since it has been reported (1) that PCNB applied to beds after "recasing" did not affect production.

Thus the inhibitory effect of PCNB appears to be rather specific in that it seems only to inhibit the induction of sexual reproduction. For once fruiting bodies (mushrooms) have been produced, this inhibitory effect is most difficult to detect. Our results from 2 years' experiments with PCNB suggest rather strongly that this chemical, if applied after harvest of the first break, at concentrations as high as 1000 ppm, does not affect mushroom production deleteriously.

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REPRODUCTION OF VARIOUS MELOIDOGYNE SPECIES IN ONION

G. D. Lewis, W. F. Mai, and A. G. Newhall[1]

Summary

Onion roots were inoculated with Meloidogyne hapla, M. incognita, M. incognita acrita, M. javanica, M. arenaria, and M. arenaria thamesi. All six species were able to cause knots on the roots. All six species were able to carry through two generations in onion roots, but the rate of reproduction in the case of M. arenaria thamesi was observed to be much lower than that of the other five species.

INTRODUCTION

Sasser[2] and others have reported on the ability of various Meloidogyne spp. to cause knotting in onion roots. However, the writers have not found reports in which it has been demonstrated that these species can successfully infect onion for more than one generation of the pathogen. Sasser reported that M. hapla, M. incognita, M. incognita acrita, M. javanica, and M. arenaria were able to cause knots in onion roots but did not test to determine whether nematodes that developed in onion roots could infect onion roots and reproduce in them. The experiments described were conducted in order to determine whether the larvae of the species used by Sasser, plus M. arenaria thamesi, are able to reproduce for two generations in onion roots.

Root knot of onion has been present in New York muckland onion fields for many years. Although M. hapla is known to cause knots in the roots of onion, it was thought for many years that the pathogen was unable to reproduce in this suscept in New York. However, knotted onion roots were found in which mature females and eggs were present. A recent survey of crop plants and weeds from the New York muckland areas indicated that M. hapla is the only root knot species present. Species determinations were made both on the basis of plant indicator tests and morphological characters. It has been observed that the severe infestations of this nematode in onion fields appear to be near the ditchbanks, especially where soil dug from the ditches has been thrown into the fields, or in weedy areas of the field. Infected weeds have been found in both locations. It is recognized that weed growth in a portion of a field sometimes occurs because of poor crop growth resulting from a nematode infestation.

EXPERIMENT I

Materials and Methods: Fourteen buckets with holes in the bottoms for drainage were steam sterilized and filled to approximately two-thirds of their depth with sterilized soil. Twelve of the buckets were inoculated, in pairs, with the six species of root knot nematode, by mixing approximately one liter of infested soil into the sterilized soil to a depth of several inches. Approximately one liter of sterilized soil was added to each of two check buckets. In each bucket, five onion sets of the yellow globe type were placed on the soil and covered with about one liter of sterilized soil. The species used in the experiment were Meloidogyne hapla, M. incognita, M. incognita acrita, M. javanica, M. arenaria, and M. arenaria thamesi.

All six species were taken from cultures obtained from the Section of Nematology, United States Department of Agriculture, and maintained in the greenhouse at Cornell. The inoculum consisted of infested soil and infected tomato roots in which the nematodes had been grown. After planting, the buckets were placed on crockery supports on a greenhouse bench and watered with care to prevent the transfer of nematodes from bucket to bucket.

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[1] Fellow, Professor, and Professor, respectively, Department of Plant Pathology, Cornell University, Ithaca, New York.

[2] Sasser, J. N. 1954. Identification and host parasite relationships of certain root-knot nematodes (Meloidogyne spp.) University of Maryland Bulletin A77.

The onion plants were allowed to grow for 12 weeks. At this time the soil was removed from each bucket and the roots washed from the soil. The washed roots from each pair of buckets inoculated with the same species were placed in a separate polyethylene bag to prevent drying. A sample of roots was taken from each bag and placed in a separate vial containing lactophenol with acid fuchsin and set aside for microscopic examination.

A handful of sterilized soil was placed in each polyethylene bag and mixed with the roots therein. The contents of these bags were saved for use as inoculum in the second experiment. The root samples preserved in the lactophenol with acid fuchsin were dissected. Mature female nematodes were removed from each sample and the species identified.

Results: The roots were knotted in all the inoculated plants. There were no knots in the roots of the check plants. In each case, only the species used for inoculum was found to be present. All six species were able to produce mature females and eggs. The egg masses of M. hapla were smaller than those of the other species.

EXPERIMENT II

Materials and Methods: The fourteen buckets used in the first experiment were filled with sand. A 6-inch pot was placed in the center of the top of each sand-filled bucket. These units were then sterilized with steam. After cooling, each pot was filled halfway with sterilized soil. The contents of each of the six polyethylene bags saved from the first experiment were divided equally and used to inoculate two of the pots. The inoculum was mixed with the soil in the pot. Two check pots received only sterilized soil. Precautions were taken to insure that only one species was placed in each pot. Each pot was then filled with more sterilized soil and planted with three 5-week-old seedlings of the onion hybrid Y-42, a yellow globe type. The location and watering of the buckets was the same as in the first experiment.

After 10 weeks the roots were washed from the soil and samples were placed in vials containing lactophenol with acid fuchsin as in the first experiment. The preserved roots were dissected and the mature female nematodes present were examined to determine the species present.

Results: The roots were knotted in all the inoculated plants. The plants inoculated with Meloidogyne arenaria thamesi had fewer knotted roots than the plants inoculated with the other species. In each case, only the species used for inoculum was found to be present. Mature females and eggs were produced by all six species. However, mature females of M. arenaria thamesi were conspicuously fewer than for the other five species. The egg masses of M. arenaria thamesi and M. hapla were small. Those of M. hapla were about the same size as they were in the first experiment.

DISCUSSION

The experiments described agree with the work of Sasser and others and add Meloigodyne arenaria thamesi to the list of species able to cause root knot symptoms in onion. It appears that M. hapla, M. incognita, M. incognita acrita, M. javanica, and M. arenaria are better able to reproduce in onion roots than is M. arenaria thamesi.

The inoculum of M. hapla used in the experiments was not isolated from onion, nor from New York onion soils, and yet it was able to reproduce in onion. Thus it appears likely that the M. hapla found in onion farm soils does not represent a special strain of this species merely because of its ability to reproduce in onion. Field experiments in which onions are grown for several seasons in the absence of other plants will be needed to demonstrate whether the M. hapla found in onion soils in New York State can increase in onion roots to a level at which subsequent onion crops are damaged, or whether damage to onions occurs only after a build-up on plants more susceptible than onion.

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EFFECTIVENESS OF DI-ELECTRIC HEAT IN KILLING ENCYSTED GOLDEN NEMATODE LARVAE

W. F. Mai

Heterodera rostochiensis Wr., the cyst-forming nematode causing the golden nematode disease of potatoes, may be transported on burlap bags. In commerce large numbers of used burlap bags are transported from farm to farm, state to state, and country to country. The use of infested bags as picking bags by a potato grower may result in the introduction of cysts of the golden nematode into field soil, storages, and equipment. Therefore, practical treatments for killing the golden nematodes adhering to burlap are of value in preventing the spread of this organism.

Fumigation of burlap bags and machinery with methyl bromide at the rate of 23 pounds per 1000 cubic feet under a gas-proof cover will kill all golden nematodes present (Lear and Mai, 1952). This treatment has been used on Long Island, New York, to help prevent spread of the golden nematode.

A number of investigators have shown that encysted larvae of the golden nematode are killed by immersion in hot water at 130°F for 5 minutes. It is usually not practical to immerse bags in hot water or treat them with steam because of the weakening of the burlap and the difficulties involved in drying.

Organisms pathogenic to farm animals have been shown to be destroyed on baled burlap bags by di-electric heat (Jungherr, 1950). The deep and rapid penetration of di-electric heat permits uniform heating of the bales without burning the outer layer of bags.

This method of disinfesting burlap feed bags is now being used commercially by the Eastern States Cooperative at their Buffalo feed mill (Flack, 1949). Because used bags which might be employed as potato picking bags were being treated commercially by di-electric heat, it was decided to test the efficacy of this treatment in killing encysted golden nematode larvae.

EXPERIMENTAL

In the first experiment 40 0.1-gram lots of cyst material (containing cysts plus bits of organic matter and seeds) were weighed. Twenty lots were placed in small nylon bags and 20 were placed in cotton bags. Four nylon and four cotton bags were placed in the center of each of four 250-bag bales of burlap bags. Four nylon and four cotton bags served as untreated checks. The bales passed under huge electrodes for a period of 55 minutes at the rate of 5 inches per minute. The temperature in the center of the bales was 230 F immediately after the bags emerged from under the electrodes. It was not possible to measure the temperature of the bags while they were passing under the electrodes.

Two weeks after treatment the cysts from each bag were exposed separately to potato root leachings. Exposure to leachings of treated cysts was repeated at 60-day intervals for a period of 6 months.

No active larvae emerged from any of the treated cysts as a result of any of the exposures to root leachings. Several thousand larvae emerged from each bag of the untreated cysts 2 weeks after treatment. The untreated cysts were discarded following the single exposure to leachings.

In the second experiment one nylon bag containing several hundred cysts was placed in the top, one in the upper middle, one in the lower middle, and one at the bottom of each of four 250-bag bales of burlap bags. Eight untreated bags served as checks.

The bales of bags were treated in the same manner as in the first experiment except that the machine was adjusted so that the temperature at the center of the bales was 205°F as the bales emerged from under the electrodes. Results were similar to those obtained from the first experiment in that no larvae emerged from the treated cysts, while large numbers emerged from the untreated ones. Six months after treatment all treated cysts were thoroughly mixed and divided into two approximately equal lots. Each lot was added to a pot of sterilized soil into which a potato plant was transplanted. No immature females developed on the roots of these plants, while substantial numbers developed on untreated checks.

DISCUSSION

Each of the two ovens at the Eastern States Feed Mill in Buffalo, N. Y. is 44 feet long, 9 feet high, and 9 feet wide. Fifty-five thousand bags can be treated with the two ovens in 16 hours at a cost of 0.5 cents per bag, which includes investment, labor, and electric current. Each oven, manufactured by General Electric Company, cost $90,000 approximately 8 years ago. In general, because of the high cost, it would not be practical to purchase dielectric equipment for use in killing nematodes adhering to burlap. However, the data presented show that this type of heat may be used to obtain a 100 percent kill of encysted golden nematode larvae present when suitable equipment is available.

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THERMOTHERAPY FOR NEMATODES OF ORNAMENTAL PLANTS

Wray Birchfield and H. M. van Pelt[1]

Summary

Spiral nematodes, Helicotylenchus multicinctus, were destroyed when subjected to 2 1/2 minutes' exposure to 50° C in water in glass vials; root-knot nematodes, Meloidogyne incognita, were destroyed at about 4 minutes' exposure to this temperature under the same condition. A high degree of control of M. incognita was realized when several different ornamental plants, infected with the nematodes, were subjected to hot-water treatment (10 minutes at 50° C). The tolerance of 24 ornamental plants to 10 minutes' exposure at 50° C (a bareroot treatment) is herein recorded.

To the Danish schoolmaster and plant pathologist Jens Ludwig Jensen, we are indebted for early information on the differential-thermal sterilization of plant material to destroy disease-producing organisms. His work with the thermal destruction of the late-blight organism in seed potatoes (23), as well as smut on oats and barley (24), formulated our present basic concepts of thermotherapy as applied to the science of plant pathology. Since Jensen's work, thermal sterilization has been used, not only against phytoparasitic fungi and bacteria, but also nematode and virus pests as well. Among the first workers to use thermotherapy for nematode diseases of plants were Cobb (12, 13), Christie and Crossman (10, 11), Chitwood (9) and Sherman (27), Hodson (22), and Staniland (28). More recently many workers have employed this type of control against phytoparasitic nematodes. A few of the many important references on this subject have been set forth in this paper for the convenience of those interested.

The State Plant Board of Florida in cooperation with the nursery industry of Florida has encouraged the production of high quality ornamental plants. This is not only desirable from the standpoint of quarantine and regulatory operations against destructive pests, but is also efficient business practice. In order to improve this program, tests with thermotherapy were established with the object of finding a means for providing the grower with relatively nematode-free propagating stock, and subsequently a more desirable product. A survey of the ornamental-growing area of Apopka, where a large volume of Florida's ornamental plants is grown, showed several important plant-parasitic nematodes, as expected. Among them were Meloidogyne incognita, Meloidogyne incognita acrita, M. arenaria, M. javanica, Pratylenchus brachyurus, and Helicotylenchus multicinctus. Meloidogyne incognita, one of the root-knot forms, and the spiral nematode, Helicotylenchus multicinctus, were most prevalent; therefore efforts at hot-water therapy were primarily directed against these. Information in the literature relative to the thermal death points of various nematodes on various plants was used as a guide for application to the particular problem.

MATERIALS AND METHODS

Meloidogyne incognita and Helicotylenchus multicinctus were subjected to 50° C (plus or minus 0.2° C) for various time intervals under two different conditions. These were: a) treatment of the free-swimming nematodes in glass vials, and b) treatment of bare-rooted plants associated with these organisms.

Glass vials, blown to a desired thinness of about 1/25 mm, and containing nematodes to be treated, were placed in a thermostatically-controlled hot-water bath (50° C). H. multicinctus was exposed for 1/2, 2 1/2, 5, and 10 minutes; M. incognita for 1, 2, 3, 4, 5, 10, and 15 minutes. Nematodes without treatment and nematodes exposed to the lag period (time necessary for the temperature to reach 50° C inside vials) were also observed. Treated nematodes, including the necessary controls, were placed on roots of a susceptible tomato variety (Rutgers) to assay the effectiveness of the treatment. After about 9 weeks the tomato plants were examined for nematodes that survived the treatment and/or galls indicating their

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[1]Phytonematologist, and Nursery Inspector, respectively, State Plant Board of Florida.

presence. Tests were replicated four to six times. Indicator plants were placed so as to avoid contamination, and two successive crops were grown.

Bare-rooted ornamental plants, in bundles of ten, were exposed 10 minutes at 50° C in a commercial-sized treating tank equipped with a mechanical agitator and thermostat (Fig. 1). Temperature was recorded with a laboratory thermometer certified by the Bureau of Standards. A record of the treatments was taken with a thermograph mounted on the tank. Treated and check plants were planted in peat that had been steamed 2 hours at 180° F, and placed on newly constructed wooden lath-house benches, either in clay pots or directly in the peat. A susceptible tomato variety was planted next to the treated plants in the peat, and later examined for gall formation to determine the effects of the heat treatment.

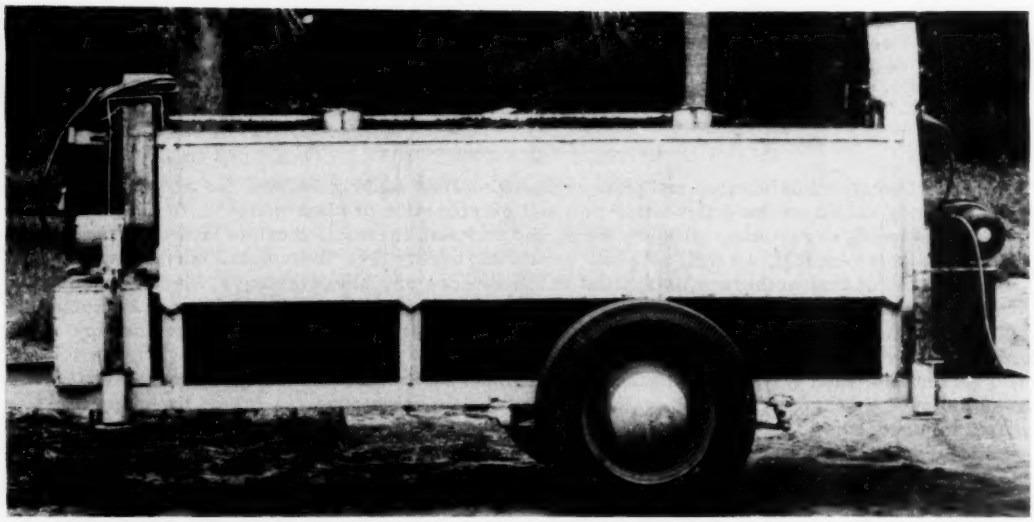

FIGURE 1. A commercial sized treating tank (hot water) equipped with mechanical agitator and thermostat for treating nematode-infected ornamental plants.

Attempts to establish two tomato plants near each treated plant were not always successful, but a comparison of the number of galled plants among treated and check plants gave a reasonable indication of the results of treatment.

EXPERIMENTAL RESULTS

When free-swimming nematodes were exposed in vials, survivors were recovered from the checks (no treatment), lag period, and 1/2-minute exposure to 50° C, but not from longer exposures in tests using Helicotylenchus multicinctus. Galls were found in the check, lag period, 1-, 2-, and 3-minute treatments but not in the 4-, 5-, 10-, or 15-minute exposures in the M. incognita tests. These results were confirmed upon examination of a second tomato crop planted in the pots.

In the case of tests made with bare-rooted plants, treated for 10 minutes at 50° C, only six out of 300 tomato plants used in the bioassay showed a few small galls compared with 83 out of 282 checks which were moderately galled. Some of the test plants thought to have been infected evidently were not; however, notes on their tolerance to the treatment were taken.

The kinds of ornamental plants tested and their tolerance to hot-water treatment, and the effects of the treatment on gall formation as determined by tomato bioassay are recorded in Table 1. Some species showed 100 percent survival without obvious injury, others were competely destroyed, while various degrees of tolerance between these extremes were observed. Succulent herbaceous types were generally damaged most; however, Gardenia and Buxus, both woody perennials, suffered a high mortality. The larger sized plants were more resistant to treatment, in general. Sansevieria spp., Aglaonema modestum, Philodendron

Table 1. List of ornamentals subjected to hot-water treatment (10 minutes at 50° C), and the effects of the treatment on the plants and on rootknot infecting their roots.

Test Plants	Survival %		Galls on Tomato	
	Check[a]	Treated	Check	Treated[b]
Aglaonema modestum Schoff.	100[1]	100	-	-
Ardesia crenata Sims	10[4]	35	+	-
Buxus sempervirens L.	100[2]	60	+	+
Caladium bicolor Vent. (8 var.)	100[1]	100	+	+
Crassula arborescens Willd.	90[2]	70	+	+
*Cryptanthus sp.	90[1]	100	+	-
Dracaena sanderiana Sander.	100[1]	100	+	+
Dracaena godseffiana Sander.	100[1]	100[1]	-	-
Gardenia jasminoides Ellis.	85[4]	35	+	+
Hibiscus rosa-sinensis L.				
*Var. American beauty ?	60[1]	10	+	-
*Var. Yellow dwarf ?	85	10	+	-
Ligustrum japonicum Thunb.	97[2]	15	+	+
Maranta leuconeura Morr. (var. Kerchoveana Morr.)	100[1]	0	+	-
*Nephthytis liberica ?				
Var. Trileaf wonder	100[2]	100	+	-
Peperomia obtusifolia Dietr.	100[1]	10	+	-
P. sandersii DC.				
(var. argyreia Bailey)	100[1]	0	-	-
*Philodendron imbe ? (canes)	100[1]	100[1]	+	-
*P. hastatum ? (leafy)	100[1]	100	+	-
*P. hastatum ? (canes)	100[1]	100	+	-
Pilea involucrata Urban	100[1]	30	+	-
Sansevieria zeylanica Willd.	90[2]	100	+	+
Sansevieria zeylanica Willd.				
Var. laurentii N.E. Br.	60[1]	50	+	-
Scindapsus aureus Engler	85[2]	15	+	-
Var. wilcoxii Hort.	100	10	+	-

[a]From 1 to 4 tests were made, as noted.
[b]Treated plants marked positive had very few galls but checks were generally moderately galled.
*Validity of name not sufficiently investigated.

imbe, P. hastatum (canes and leafy), and Caladium bicolor tolerated the treatment.

COMMERCIAL TREATMENT

A commercial-sized tank was constructed for treating plants in large numbers for the industry (Fig. 1). Several million Sansevieria roots were treated during the 1956 and 1957 seasons. Growers were satisfied with growth response in treated plants. Other ornamentals which were satisfactorily treated on a commercial scale were Philodendron and Chinese evergreen. Ten minutes' exposure at 50° C did not give satisfactory control of rootknot on Caladium. Additional greenhouse tests showed that Caladium bicolor (several horticultural varieties) withstood 20 minutes' exposure to 50° C. Preliminary tests indicated that 15 minutes at 50° plus a 1:100 solution of commercial formalin (40% formaldehyde) might give satisfactory root-knot control on this plant.

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STATE PLANT BOARD OF FLORIDA, GAINESVILLE, FLORIDA

ADDITIONS TO THE HOST LISTS OF THE NEMATODES PARATYLENCHUS PROJECTUS AND TRICHODORUS CHRISTIEI[1]

B. W. Coursen[2], R. A. Rohde[2], and W. R. Jenkins[3]

Abstract

Of 101 plant species and varieties tested, 89 were found to be hosts of the pin nematode, Paratylenchus projectus. The known host range of the stubby-root nematode, Trichodorus christiei, was extended with the addition of 66 more plant species and varieties.

The pin nematode, Paratylenchus projectus, was described from roots of pasture grasses in Maryland (3). Populations of this species were subsequently maintained in greenhouse cultures on tall fescue grass and tobacco. No other hosts have been reported.

A partial host list of a species of Trichodorus was reported in 1957 (4). Since then this species has been identified as T. christiei (1).

Additional plants were tested as possible hosts of each of these two species of nematodes. Inoculations were made under greenhouse conditions on plants growing in 4- or 6-inch pots. Each pot was infested with 100 specimens of either P. projectus or T. christiei. After 50 to 60 days, nematodes were removed from the soil by the method of Christie and Perry (2) and population size determined.

Since some individuals of P. projectus will persist in sterile soil for more than 60 days, the mere presence of nematodes after this period was not sufficient for determining hosts. Therefore, those plants on which the final number was less than the initial inoculum level of 100 were listed as non-hosts.

It has been determined that T. christiei will not persist in sterile soil for 60 days (4); therefore, recovery of individuals after this period of time was considered as evidence that the nematodes were reproducing on this plant.

A total of 101 plant species and varieties representing 10 families were tested as possible hosts of P. projectus. Only 12 of these plants were non-hosts. The results of these tests are given in Table 1.

Table 1. Reaction of plants as hosts or non-hosts to Paratylenchus projectus in greenhouse tests.

Family and Scientific Name	Common Name and Horticultural Variety
NON-HOSTS	
Chenopodiaceae:	
Spinacia oleracea L.	Spinach: American
Compositae:	
Cichorium endivia L.	Endive: Green Curled
Cucurbitaceae:	
Cucumis sativus L.	Cucumber: Marketeer, Palmetto
Cucurbita pepo var. melopepo Alef.	Squash: Caserta

[1]Scientific Article No. A678, Contribution No. 2893, of the University of Maryland Agricultural Experiment Station, Department of Botany.
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Table 1. (Continued).

HOSTS	
Gramineae:	
Trifolium hybridum L.	Alsike Clover
Solanaceae:	
Capsicum frutescens var. *grossum* Bailey	Sweet Pepper: California Wonder, Early Calwonder, Yolo Wonder
Lycopersicon esculentum var. *commune* Bailey	Common Tomato: Rutgers
Solanum melongena var. *esculentum* Nees.	Common Eggplant: Black Beauty, Florida High-Bush
Umbelliferae:	
Daucus carota var. *sativa* DC.	Cultivated Carrot: Red-Core, Gold Pak Chantenay
Petroselinum crispum Nym.	Parsley: Market Gardener's Best, Triple Curled
Chenopodiaceae:	
Beta vulgaris L.	Beet: Mammouth Long Red, Winterkeeper, Perfect Detroit Dark Red
Beta vulgaris var. *cicla* L.	Swiss Chard: Fordhook Giant
Spinacia oleracea L.	Spinach: Long-standing Bloomsdale Savoy
Compositae:	
Lactuca sativa L.	Lettuce: Great Lakes, Slobolt
Cruciferae:	
Brassica caulorapa Pasq.	Kohlrabi: Early White Vienna
Brassica fimbriata DC.	Kale: Dwarf Blue Curled Scotch, Dwarf Green Curled Scotch
Brassica oleracea var. *botrytis* L.	Cauliflower: Early Snowball
Brassica oleracea var. *capitata*	Cabbage: Bonanza, Golden Acre, Jersey Wakefield, Marion Market, Wisconsin All-Season
Brassica oleracea var. *gemmifera* Zenker	Brussels Sprouts: Perfection
Brassica oleracea var. *italica*	Broccoli: De Cicco, Italian Green Sprouting
Brassica rapa L.	Turnip: Purple-top White Globe
Brassica sp. L.	Mustard: Fordhook Fancy
Raphanus sativus L.	Radish: Cherry Belle, White Icicle
Raphanus sativus var. *longipinnatus* Bailey	Radish: China Rose
Cucurbitaceae:	
Cucurbita pepo var. *melopepo* Alef.	Squash: Early Prolific Straightneck
Gramineae:	
Avena sativa L.	Oat: Arlington, Atlantic, Clinton 59, Lee Cold Proof
Bromus inermis Leyss.	Brome-Grass
Dactylis glomerata L.	Orchard Grass

Table 1. (Concluded).

Festuca elatior L.	Tall Fescue: Kentucky 31
Festuca rubra L.	Red Fescue
Hordeum vulgare L.	Barley: Kenbar, Wong
Pennisetum glaucum R. Br.	Pearl Millet: Common, Star
Phleum pratense L.	Timothy: Ill Grown
Poa pratensis L.	Kentucky Blue-Grass: Common, Merion
Secale cereale L.	Rye: Abruzzi, Balbo, Tetra Petkus
Sorghum vulgare var. *sudanense* Hitchc.	Sudan-Grass
Triticum aestivum L.	Wheat: Leapland, Tayland, Thorne
Zea Mays L.	Field Corn: Conn. 870, Pa. 602, U.S. 131, U. S. 505, U. S. 578
Zea Mays var. *rugosa* Bonaf.	Sweet Corn: Aristogold, Bantam Evergreen, Country Gentleman, Golden Bantam, Golden Cross Bantam, Iochief
Leguminosae:	
Glycine Max Merr.	Soybean: Dorman, John Kolk Clark, Lee, Ogden
Lespedeza sp. Michx.	Lespedeza: Common Korean, Climax Korean, Kobe, Rowan Korean
Medicago sativa L.	Alfalfa: Buffalo, Kansas Common, Williamsburg
Melilotus alba Desr.	White Sweet Clover
Trifolium incarnatum L.	Crimson Clover
Trifolium pratense L.	Red Clover: Kenland, Local, Medium
Trifolium repens L.	White Clover: Ladino
Vicia villosa Roth.	Hairy Vetch
Liliaceae:	
Allium cepa L.	Onion: Beltsville Bunching, Sweet Spanish
Malvaceae:	
Hibiscus esculentus L.	Okra: Dwarf Green Prolific, Emerald Green Velvet
Solanaceae:	
Nicotiana tabacum L.	Tobacco: Catterton
Lycopersicon esculentum var. *commune* Bailey	Common Tomato: Beefsteak, Big Boy, Chesapeake, Sunray
Umbelliferae:	
Apium graveolens var. *dulce* Pers.	Celery: Giant Pascal, Golden Self-Blanching

Sixty-six plant species and varieties representing 10 familes were added to the host list of *T. christiei*. These results are presented in Table 2.

Table 2. Hosts of *Trichodorus christiei* as determined by greenhouse tests.

Family and Scientific Name	Common Name and Horticultural Variety
Chenopodiaceae:	
Beta vulgaris	Beet: Mommouth Long Red, Winterkeeper
Beta vulgaris var. *cicla* L.	Swiss Chard: Fordhook Giant
Compositae:	
Cichorium endivia L.	Endive: Green Curled
Lactuca sativa L.	Lettuce: Slobolt
Cruciferae:	
Brassica caulorapa Pasq.	Kohlrabi: Early White Vienna
Brassica fimbriata DC.	Kale: Dwarf Blue Curled Scotch Dwarf Green Curled Scotch
Brassica oleracea var. *botrytis* L.	Cauliflower: Early Snowball
Brassica oleracea var. *capitata*	Cabbage: Bonanza, Jersey Wakefield, Marion Market, Wisconsin All-Season
Brassica oleracea var. *gemmifera* Zenker	Brussels Sprouts: Perfection
Brassica oleracea var. *italica*	Broccoli: Di Cicco, Italian Green Sprouting
Brassica sp. L.	Mustard: Fordhook Fancy
Raphanus sativus L.	Radish: Cherry Belle, White Icicle
Raphanus sativus var. *longipinnatus* Bailey	Radish: China Rose
Cucurbitaceae:	
Cucumis melo L.	Muskmelon: Honeydew
Cucurbita pepo var. *melopepo* Alef.	Squash: Caserta, Early Prolific Straightneck
Gramineae:	
Festuca rubra L.	Red Fescue
Hordeum vulgare L.	Barley: Kenbar, Wong
Pennisetum glaucum R. Br.	Pearl Millet: Common, Star
Phleum pratense L.	Timothy: Ill Grown
Triticum aestivum L.	Wheat: Leapland, Tayland, Thorne
Zea Mays L.	Field Corn: Conn. 870, Pa. 602, U.S. 505, U.S. 578
Zea Mays var. *rugosa* Bonaf.	Sweet Corn: Aristogold, Bantam Evergreen, Country Gentleman, Golden Bantam, Golden Cross Bantam, Iochief
Leguminosae:	
Arachis hypogea L.	Peanut: Virginia Jumbo Runner
Glycine Max Merr.	Soybean: Dorman, John Kolk Clark, Ogden
Lespedeza sp. Michz.	Lespedeza: Common Korean, Kobe, Rowan Korean
Medicago sativa L.	Alfalfa: Buffalo, Kansas Common
Trifolium pratense L.	Red Clover: Kenland, Medium

Table 2. (Concluded).

Liliaceae:		
Allium cepa L.	Onion:	Beltsville Bunching, Sweet Spanish
Malvaceae:		
Hibiscus esculentus L.	Okra:	Emerald Green Velvet
Solanaceae:		
Capsicum frutescens var. *grossum* Bailey	Sweet Pepper:	California Wonder, Early Calwonder, Yolo Wonder
Lycopersicon esculentum var. *commune* Bailey	Common Tomato:	Big Boy, Rutgers, Sunray
Solanum melongena var. *esculentum* Nees.	Common Eggplant:	Black Beauty, Florida High-Bush
Umbelliferae:		
Apium graveolens var. *dulce* Pers.	Celery:	Giant Pascal, Golden Self-Blanching

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UNIVERSITY OF MARYLAND AGRICULTURAL EXPERIMENT STATION, DEPARTMENT OF BOTANY

THE BURROWING NEMATODE, RADOPHOLUS SIMILIS, IN ROOTS OF CROTALARIA SPECTABILIS

Harry W. Ford and Chancellor I. Hannon[1]

The burrowing nematode, Radopholus similis (Cobb) Thorne, an endoparasite, is the primary cause of a serious citrus disease known as spreading decline (6). The only satisfactory method of control at present is to remove infected trees, together with a border of four rows of adjacent healthy trees, and fumigate the area with dichloropropene-dichloropropane mixture. An elaborate containment program, under the direction of the Florida State Plant Board, utilizing the "pull and treat" procedure, has been in progress for several years (3). This program includes the recommendation that no host plants be permitted to grow on the treated land during the 2-year waiting period before replanting to citrus, as a precaution against failure of fumigation. Use of a non-host cover-crop on such areas would be desirable in preference to fallow fields. When roots of Crotalaria spectabilis Roth., grown in infested field soil in a temperature tank, 74-78° F, were examined, using the Young procedure (9), no nematodes were recovered. Thus, C. spectabilis has been recommended as a non-host cover-crop (7).

The method by which C. spectabilis was judged to be a non-host of the burrowing nematode did not give specific information about the penetration of the nematode into roots, its subsequent ability to lay eggs, or the extent of any damage within the roots. It is the purpose of this paper to report that R. similis is capable of penetrating roots of C. spectabilis and laying viable eggs therein, also to point out that C. spectabilis is a poor host plant for R. similis, although the nematode may complete a life cycle in its roots. In a control program which seeks 100 percent eradication of the burrowing nematode from commercial citrus areas, C. spectabilis, by virtue of its uncertain host status, has doubtful value as a cover crop.

METHODS AND RESULTS

The technique used in these studies is as follows: 2-day-old seedlings of C. spectabilis were planted in Petri dishes filled with sterile fine sandy soil and covered with an aluminum lid to eliminate light and retard drying. Two seedlings were planted in each Petri dish. The root tip of each plant was laid on a moist rectangle of Whatman No. 41 filter paper, the center of which had been sprinkled lightly with sand. A 3/4-inch cube of wood, previously soaked in water, was placed over the root tip and the root and block were surrounded with soil. To inoculate a root tip, the block was removed and 25 female burrowing nematodes were placed on each root with a micropipette (1). The filter paper, in addition to reducing downward migration of the nematodes, served to identify the area of inoculation when the plants were eventually removed from the dish for staining. Roots were stained by the aceto-osmium procedure (9) to determine whether the nematodes had entered the roots, what tissues were invaded, and the number of eggs deposited.

In a preliminary study, 20 2-day-old C. spectabilis plants were inoculated and held for 5 days. It was found that 80 to 100 percent of the nematodes penetrated the roots, resulting in extensive necrosis in the root cortex. Eggs were found in two of the roots. When the stained nematodes in the roots were examined under the compound microscope, the body contents appeared to be coagulated. In contrast, when roots of seedling rough lemon (Citrus limon) were infested for 5 days, only 20 to 70 percent of the nematodes entered the roots and laid 2 to 10 eggs per nematode. The nematodes were stained by the osmium and showed little or no evidence of coagulation of intestinal contents.

The following experiment was carried out to determine the ability of burrowing nematodes in Crotalaria to infest other plants. Twenty 2-day-old C. spectabilis seedlings were inoculated, divided into five groups of four plants each, and held for 2, 4, 6, 10, and 17 days respectively. The infested roots of two plants in each group were diced with scissors and placed on the root-tips of two C. spectabilis plants; the two roots remaining were chopped and placed on root-tips of okra, a highly susceptible host of R. similis (2). Each set of bio-

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assayed roots was examined after 5 days. No nematodes were found in either the Crotalaria or the okra roots.

Thirty-two inoculated C. spectabilis seedlings were used to determine survival of burrowing nematodes in the roots and development of larvae from the few eggs deposited. Examinations were made at 3, 4, 5, 10, 15, 20, 25, and 30 days from inoculation. At each date, four plants were removed and their roots dissected. From the results (Table 1) and previous knowledge of penetration of C. spectabilis roots by the nematode, it is assumed that not all the nematodes that penetrated the roots were recovered by dissection. The nematodes that were found appeared emaciated and were extremely sluggish, even when placed in 2% hydrogen peroxide. All roots held for 10 days or longer had visible decaying tissue in the region of nematode activity.

Table 1. Burrowing nematodes dissected from the roots of Crotalaria spectabilis after different periods of incubation.

Length of Infestation (Days)	Number of nemas dissected from four roots.[a]			
	Dead		Alive	
3	26 females		10 females	
4	16 females		20 females	
5	21 females		8 females	
10	9 females		8 females	
15	8 females		20 females	14 larvae
20	16 females	10 larvae	4 females	
25	15 females	8 larvae	0	
30	4 females	3 larvae	2 females	1 larva[b]

[a] 25 female burrowing nematodes had originally been placed on each root in Petri dishes.

[b] The larva was in the fourth stage.

DISCUSSION AND CONCLUSIONS

The nematode resistance feature of Crotalaria spectabilis has been of interest because of the reaction of Crotalaria to the root-knot nematode. The root-knot nematode is unable to complete a life cycle in the roots of Crotalaria. McBeth and Taylor (5) reported that larvae of root-knot nematodes, Meloidogyne spp., entered the roots of C. spectabilis but did not develop beyond the fourth larval stage. Martin (4) found a few females of Meloidogyne javanica (Treub.) Chitwood, after examining many roots of C. spectabilis, but no egg laying was observed.

The appearance and activity of Radopholus similis inside the roots of C. spectabilis suggest that this plant, although susceptible to root damage, is not a favorable host for the burrowing nematode. However, the fact that larvae were observed in inoculated roots indicates that viable eggs were produced and hatched. Bioassay of inoculated roots indicated that the females from the original inoculum were incapable of causing further infections.

Data in Table 1 indicate considerable variation in the ability of individuals of a population of R. similis to survive on roots of C. spectabilis. Such variability is to be expected in a biological population. It has not been established whether the females obtained from roots 30 days after inoculation belong to the second generation, thus there is a degree of uncertainty associated with the assumed resistance of C. spectabilis to the burrowing nematode. The use of C. spectabilis as a cover-crop in a program which seeks complete eradication of the burrowing nematode is questioned.

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DAMPING-OFF OF SWEET ORANGE SEEDLINGS BY RHIZOCTONIA SOLANI CONTROLLED WITH BIPHENYL

L. J. Klotz, Po-Ping Wong, and T. A. DeWolfe

Heiberg and Ramsey found the vapor from biphenyl fungistatic to Rhizoctonia solani Kuehn growing on potato-dextrose agar. This was confirmed in the work reported here, in which inoculum of the fungus was planted at the center of the surface of potato-dextrose agar in a standard Petri dish and exposed to biphenyl vapor. Covers of the Petri dish cultures were removed and the dishes inverted on wire screen in two deep culture dishes (capacity 6785 cc), one of which had 0.5 grams and the other 5.0 grams of biphenyl. During the 48 hours exposure to the vapor of the chemical the fungus made no growth. When the cultures were removed from the vapor they resumed growth, as shown in Figure 1, indicating that

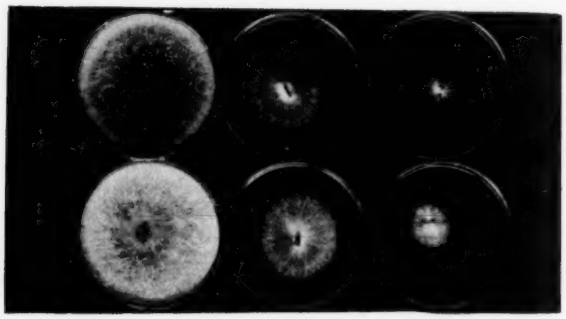

FIGURE 1. Fungistatic action of biphenyl on Rhizoctonia solani Kuehn (citrus strain). Control plates (vertical pair left, potato-dextrose agar) were covered by fungus in 48 hours. During that period at 20°C there was no growth on plates (vertical pairs in center and at right) exposed to the chemical. After removal from biphenyl vapor the fungus in 2 days made the growth shown. The cultures of the central vertical pair were exposed to 0.5 gm of biphenyl in a deep culture dish of 6785 cc capacity; those at right to 5.0 gm in same volume.

the chemical was fungistatic and not fungicidal during that period. It is seen, however, that the cultures that had been in the presence of the smaller amount (0.5 gram) of biphenyl resumed growth much more rapidly than those which had been exposed for 2 days to 5 grams.

Since this fungus is the most pathogenic of those causing damping-off of citrus seedlings we have determined the respective disease-controlling and phytotoxic effects of biphenyl on the fungus and its citrus host. To accomplish this, standard greenhouse soil mix was sterilized with chloropicrin and placed in gallon cans. The several quantities of biphenyl were sprinkled on the soil surface and worked into the first inch. Twenty-five seeds of Homosassa sweet orange were distributed on the soil in each can and pressed into the surface layer to a depth equal to the width of the seed. Inoculum of the fungus for each can to receive it was made by mixing half of a Petri dish mat on potato-dextrose agar with 100 ml. of water in a Waring Blendor. This was then poured onto the seeds and soil and covered with 3/4 inch of heat-sterilized sand. For controls, agar without the fungus was added. The moisture content of the soil was maintained at 14 to 16 percent by weighing the cans and adding water as required. The following tabulation and Figure 2 show the quantities of biphenyl used and their effect on germination, emergence, damping-off, and phytotoxicity. Phytotoxicity is shown by the relative number of seedlings.

The results show that quantities of 5 to 40 grams of biphenyl per can are effective in controlling damping-off. All amounts of the chemical used (1 to 40 grams) are somewhat toxic to the citrus seedlings, as is indicated by the reduced germination and numbers of seedlings in the absence of the fungus. Moreover, the seedlings emerged more rapidly in the absence of biphenyl.

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[1] Heiberg, Barbara C., and Glen B. Ramsey. 1946. Fungistatic action of biphenyl on some fruit and vegetable pathogens. Phytopathology 36: 887-891.

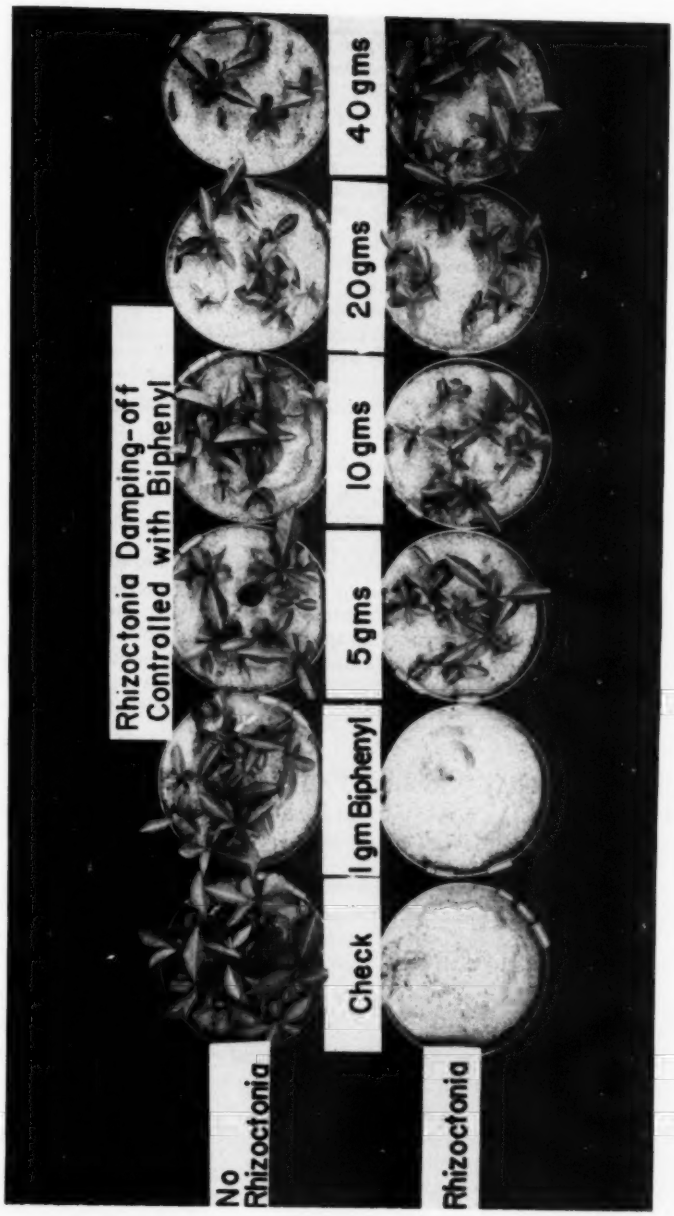


FIGURE 2. Damping-off of Homosassa sweet orange seedlings controlled with biphenyl.

Table 1. Fungitoxicity and phytotoxicity of biphenyl.

Grams Biphenyl per can	Percentage Germination of sweet citrus seed		Number of Seedlings[a] from 100 seeds[b]		Number of Seedlings damped-off (post emergence)	
	No Rhizoctonia	Rhizoctonia added	No Rhizoctonia	Rhizoctonia added	No Rhizoctonia	Rhizoctonia added
0	90	0	158	0	0	0[c]
1	72	8	116	9	0	4[d]
5	58	87	63	105	0	0
10	61	61	86	94	0	0
20	51	63	71	96	0	0
40	58	78	72	106	0	0

[a]Considerable polyembryony noted.

[b]Results from total of 4 cans, each with 25 seeds.

[c]All pre-emergence damping-off; no seedlings emerged.

[d]Of 9 seedlings that emerged 4 were damped off; no other seedlings emerged because of effect of fungus and other factors on germinability.

While the chemical is effective against Rhizoctonia solani it is, as shown by investigations by DeWolfe and Klotz (unpublished) and by Heiberg and Ramsey[1], ineffective against Phytophthora spp. that cause root rot, fruit rot, and gummosis of citrus, and against Pythium spp. that damp-off seedlings of many plants. For this reason it is not offered as a substitute for the approved procedures for controlling damping-off in citrus seed beds. On the other crops, however, where Phytophthora spp., Pythium spp., and other biphenyl-resistant fungi play no pathogenic role the chemical may be found useful for the control of damping-off.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, RIVERSIDE, CALIFORNIA

RELATIVE EFFECTS OF ETHYL THIONOCARBAMATE AND DOWICIDE A-HEXAMINE ON FOUR CITRUS FRUIT PATHOGENS

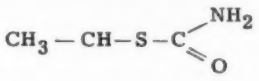
S. Z. Berry[1]

Abstract

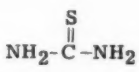
Five percent ethyl thionocarbamate, C.P. 2229, was demonstrated to be more inhibiting than 2% Dowicide A-Hexamine to orange decay produced by Diplodia natalensis, Phomopsis citri, Penicillium digitatum and Penicillium italicum. Both formulations were fungistatic to the four pathogens. In vivo neither chemical showed specificity for D. natalensis or P. citri. However, C.P. 2229 controlled decay produced by P. digitatum more effectively than that produced by P. italicum; the latter became more marked after the treated oranges were stored for 3 weeks.

In vitro the inhibition produced by C.P. 2229 on the four citrus pathogens was less than that of the commercial Dowicide A-Hexamine. Neither formulation in vitro showed specificity for D. natalensis or P. citri. However, the two chemicals were specific for Penicillium, each being more effective against P. digitatum than against P. italicum.

Ethyl thionocarbamate, henceforth called C.P. 2229[2], was shown to be of considerable promise as an inhibitor of citrus decay in storage and shipping tests from Orlando, Florida[3]. In shipping tests with treated prepackaged Florida oranges Hruschka, Winston and Lutz (2) showed it to be an effective decay-inhibiting compound. C. P. 2229



is similar to thiourea



The activity of thiourea was indicated by Childs and Siegler (1) to be based on the presence of both an amino group and a sulfur group. Both of these components are possessed by C.P. 2229.

The present study was begun to determine the effects of C.P. 2229 on the important postharvest pathogens of Florida citrus, Diplodia natalensis Pole-Evans and Phomopsis citri Fawc., which cause stem end rot, and on Penicillium digitatum Sacc., which causes green mold, as compared with the effects of commercially used Dowicide A-Hexamine called Dowicide herein.

The observation of the development of blue mold, caused by P. italicum Wehmer, on oranges treated with C.P. 2229 in spite of the control of P. digitatum, led to a study of P. italicum also. Blue mold is important as a postharvest disease on California citrus, but only occasionally is it a problem on Florida citrus; in contrast, green mold is important in both citrus areas.

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[1] Pathologist, Crops Research Division, United States Department of Agriculture. The writer wishes to express his appreciation to J. F. L. Childs for his suggestions and to G. L. Roberts for assistance in harvesting the oranges.

[2] Monsanto Chemical Company, St. Louis, Missouri.

[3] Personal correspondence with J. R. Winston.

MATERIALS AND METHODS

A 2% solution of a commercial preparation of Dowicide consisting of sodium orthophenylphenol 40%, hexamine 20%, and inert ingredients 40% was compared with a 5% solution of C.P. 2229 in all tests.

For the in vivo studies naturally as well as artificially inoculated oranges were used. The oranges were washed and dried after picking. Fruits were inoculated on the stem end with inoculum prepared by grinding up cultures of the respective stem-end rot organisms in a Waring Blendor with water and incubated in moisture chambers consisting of closed cans 7 cubic feet in volume, provided with vents and lined with moistened toweling. D. natalensis-inoculated oranges were incubated at 83° F and P. citri-inoculated ones at 70°; these respective temperatures approximate the optimum for each organism. After an incubation period of 30 hours, 60 hours, or 100 hours the fruits were removed from the chamber, washed free of inoculum, dried, and then treated with the appropriate chemical solutions. Between inoculation and treatment the fruits were stored at 70°. The control fruits were washed only before being stored.

D. natalensis and P. citri become established before harvest on the buttons of the stem of the oranges, but only after harvest do they resume activity (4). For this reason, rather long intervals were used between inoculation and treatment in the tests with the stem-end rot organisms. P. digitatum and P. italicum, on the other hand, do not attack until the oranges have been harvested and penetration occurs through wounds on the rind. Consequently, inoculations were made by dipping the oranges in a water suspension of the spores of either P. digitatum or P. italicum and then puncturing the fruits by rolling each on a row of 1/16-inch protruding points. Oranges so inoculated were then incubated in the moisture chambers at 70° F for 6 hours before treatment with the chemicals.

After immersion of the fruits in the chemical treating solutions for the specified period (3 or 6 minutes) they were rinsed in water and dried. The treated fruits were stored at 70° F. The control oranges received no treatment other than the initial washing. In five tests for stem-end rot control an average of 247 oranges was used per treatment, making a total of 5,187. Seven tests with green mold and blue mold are reported here. An average of 132 oranges per treatment, a total of 1,056 oranges, was used in the latter mold tests.

The growth-inhibiting characteristics of the compounds were assayed in potato-dextrose agar. After inoculation the plates were held at 75° F. The four pathogens studied were all isolated from diseased oranges. D. natalensis and P. citri were grown on steamed oats. In the assay tests consistent results were obtained by seeding the test plates with individual oat kernels taken from 3-week-old cultures of the organisms. P. digitatum and P. italicum were maintained on oranges and 1/4-square-inch portions of the rind of infected oranges were used for seeding the assay plates.

RESULTS AND CONCLUSIONS

Stem-End Rot

C.P. 2229 was more effective in the control of stem-end rot than Dowicide in naturally as well as artificially inoculated oranges (Table 1). After 3 weeks of storage naturally inoculated oranges treated with C.P. 2229 showed 1.0 percent stem-end rot as compared with 10.1 percent for oranges treated with Dowicide. Decay in the untreated controls was 18.3 percent. In oranges artificially inoculated with D. natalensis and treated 30 hours after inoculation with C.P. 2229 there was 1.4 percent stem-end rot after a 3-week storage period, as compared with 32.6 percent for fruit treated with Dowicide. There was 62.4 percent decay in the untreated control lots. Both chemicals acted as fungistats, but they differed somewhat in their residual effect. Thus, C.P. 2229-treated fruit did not show a large increase in decay until after 3 weeks' storage, while the Dowicide-treated oranges showed a rapid increase in stem-end rot after only 2 weeks (Table 1).

C.P. 2229 was tested further by treating oranges 60 and 100 hours after inoculation. As expected, the amount of decay increased as the period between inoculation and treatment increased (Figs. 1 and 2). This was also true for fruit treated with Dowicide. When similar tests were carried out with C.P. 2229 on naturally inoculated oranges the decay was greater on the fruit treated 60 hours after harvest than on that treated 30 hours after harvest. However, oranges treated 100 hours after harvest, contrary to expectation, showed relatively less decay than the oranges treated after 30 or 60 hours (Fig. 1). This anomalous result might be explained by the stem-end rot organisms being in a stage of development more susceptible to chemical

Table 1. Effect of time of application of C.P. 2229 and of Dowicide on the development of stem-end rot in naturally and artificially inoculated oranges[a].

Treatment[b] and inoculation	Time between harvest or inoculation and treatment (hours)	Percent fruits with stem-end rot at indicated period after inoculation[c]			
		2 weeks	3 weeks	4 weeks	5 weeks
None (control):					
Natural	--	9.8	18.3	24.8	35.4
Diplodia natalensis	--	48.4	62.4	72.9	77.0
Phomopsis citri	--	24.4	60.0	76.3	83.8
5% C.P. 2229:					
Natural	30	0.5	1.0	2.7	7.1
	60	0.8	2.3	6.6	13.6
	100	0.5	0.5	1.0	6.1
D. natalensis	30	0.5	1.4	15.8	37.0
	60	12.8	23.1	38.7	52.0
	100	17.9	31.2	41.2	53.5
P. citri	30	0.5	2.7	14.6	32.8
	60	0.5	4.4	27.5	46.4
	100	2.6	8.8	31.2	49.0
2% Dowicide:					
Natural	30	4.4	10.1	20.3	24.7
	60	1.0	9.3	19.4	27.0
	100	4.4	11.0	20.0	30.9
D. natalensis	30	11.9	32.6	54.6	70.7
	60	20.6	38.6	60.0	73.1
	100	26.2	43.4	64.1	70.6
P. citri	30	6.5	43.6	73.9	84.9
	60	6.8	47.3	68.9	81.3
	100	8.8	44.4	74.0	81.4

[a] Five tests with harvest dates (1957) as follows: Pineapple oranges, March 8; Valencia oranges, March 8; April 5, April 29, and May 27. An average of 247 oranges per each treatment (total = 5187 oranges).
[b] Dipped for 3 minutes in the solution, rinsed with water and then dried.
[c] Stored at 70° F.

inhibition 100 hours after harvest than when treatment was earlier. Another possible explanation would be changes in the host tissue, such as loss of moisture, with resultant greater effectiveness of the treating chemicals.

In vitro no specificity in the effects of C.P. 2229 (inhibitory concentration 450 to 500 ppm.) and Dowicide (40 to 60 ppm.) was observed on D. natalensis and P. citri (Table 2). Likewise, when C.P. 2229 or Dowicide was used 30 hours after inoculation on oranges artificially inoculated with these organisms no specificity was observed. However, when 100 hours elapsed before treatment, particularly when C.P. 2229 was used, the D. natalensis-inoculated oranges showed more decay than the P. citri-inoculated (Fig. 2).

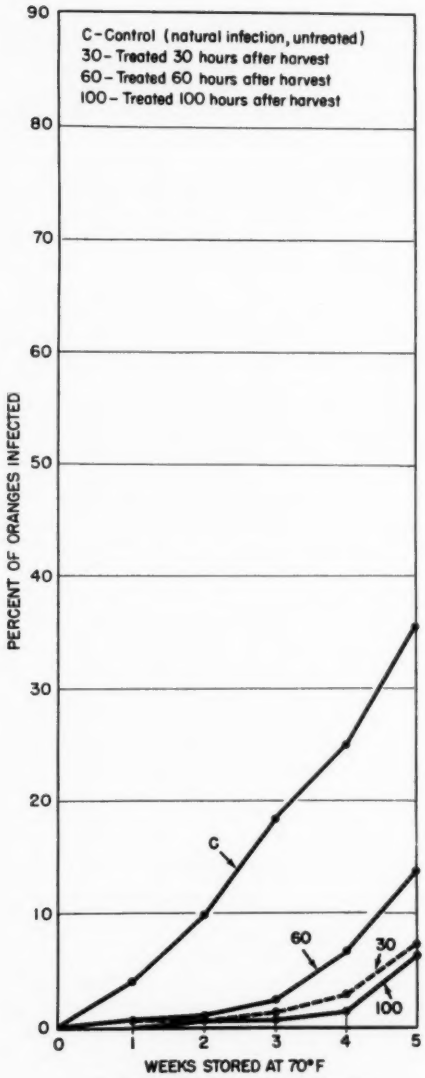

Fig. 1. The effect of C.P. 2229 on stem-end rot development in naturally infected oranges treated 30 hours, 60 hours, and 100 hours after harvest.

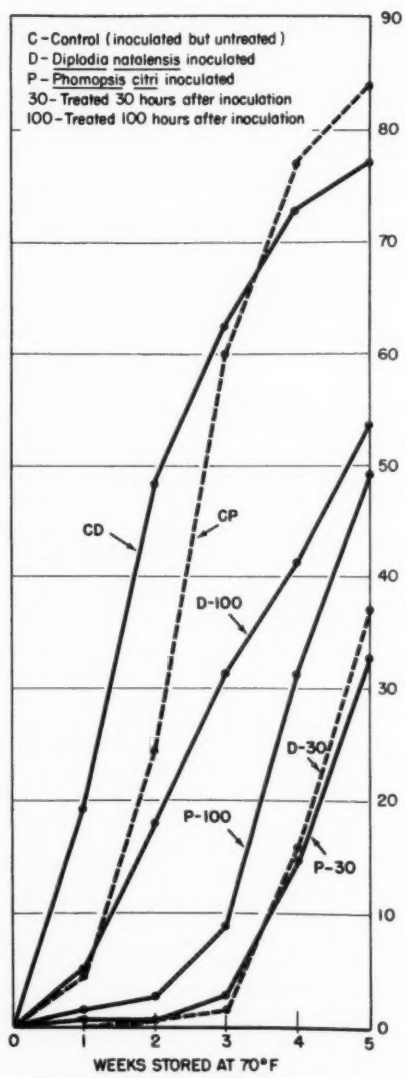

Fig. 2. The effect of C.P. 2229 on stem-end rot development in oranges artificially inoculated with D. natalensis or P. citri. Treated 30 hours and 100 hours after inoculation

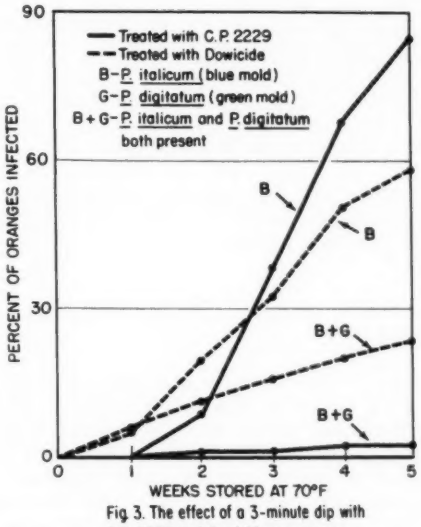

Fig. 3. The effect of a 3-minute dip with C.P. 2229 and Dowicide on oranges inoculated with P. italicum (blue mold)

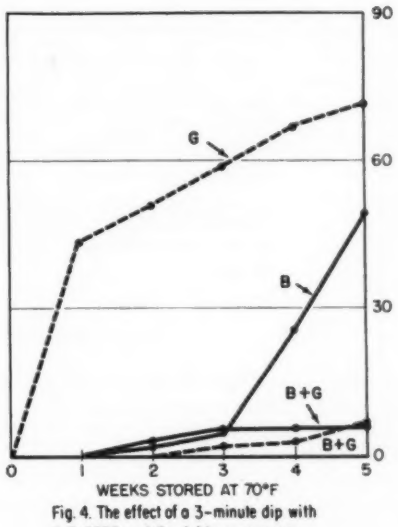

Fig. 4. The effect of a 3-minute dip with C.P. 2229 and Dowicide on oranges inoculated with P. digitatum (green mold).

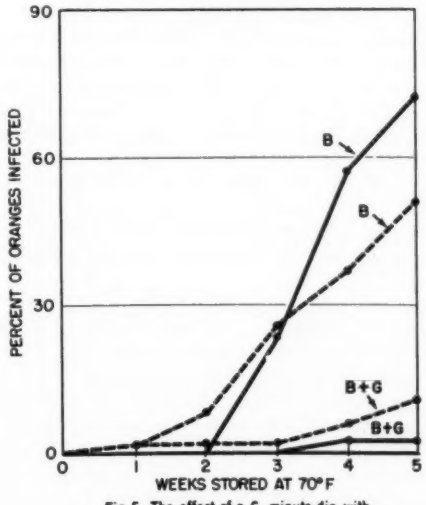

Fig. 5. The effect of a 6-minute dip with C.P. 2229 and Dowicide on oranges inoculated with P. italicum (blue mold).

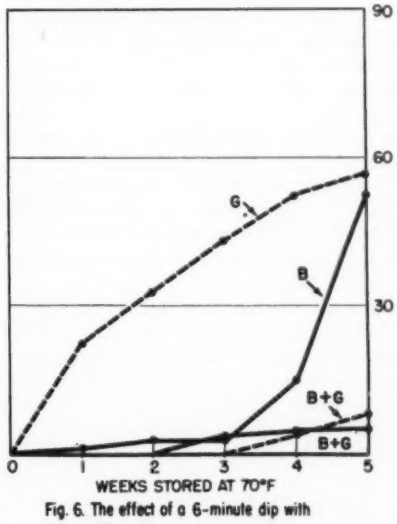

Fig. 6. The effect of a 6-minute dip with C.P. 2229 and Dowicide on oranges inoculated with P. digitatum (green mold).

Table 2. The activity in vitro of C.P. 2229 and of Dowicide[a].

Organism	Inhibitory concentration (parts per million)	
	C.P. 2229	Dowicide
Diplodia natalensis	450 to 500	40 to 60
Phomopsis citri	450 to 500	40 to 60
Penicillium digitatum	500 to 540	15 to 20
Penicillium italicum	550 to 580	70 to 80

[a] Incubated for an 8-day period at 75° F. A potato-dextrose agar medium was used as the diluent.

Green and Blue Mold Rots

The tests reported in this section were carried out to determine the effect of C.P. 2229 on blue and green molds. For oranges inoculated with P. italicum it was found that the 3-minute dip in C.P. 2229 was effective for 2 weeks in controlling blue mold. However, decay rapidly increased and after 3 weeks there was more blue mold decay on the oranges treated with C.P. 2229 than on comparative lots of fruit treated with Dowicide (Fig. 3). When the treatment time was increased to 6 minutes the results were similar; however, both C.P. 2229 and Dowicide reduced mold development more (Fig. 5). Because of wounding the fruit in the inoculation procedure a certain amount of secondary infection occurred, and some oranges showed green mold as well as blue mold. C.P. 2229 reduced the secondary infection by green mold to almost negligible proportions, but Dowicide was not as effective (Figs. 3 and 5).

In oranges artificially inoculated with the green mold fungus, C.P. 2229 completely controlled green mold alone for the 5-week storage period of the test. However, after 3 weeks blue mold appeared and then increased sharply in severity (Fig. 4). This latter test duplicated the findings on naturally inoculated fruit. Although green mold is the primary disease organism and blue mold of minor importance, such oranges when treated with C.P. 2229 show blue mold without any green mold. When oranges were dipped for 6 minutes there was more inhibition of blue mold, but the same relative trends existed (Figs. 4 and 6). Thus, C.P. 2229 demonstrated greater inhibitory action against green mold than against blue mold. However, since this action was not expressed until 3 weeks after treatment, the lack of control of blue mold by C.P. 2229 would have to be considered from the practical standpoint only in fruit to be stored more than 3 weeks.

This specificity was further tested in vitro. Marloth's (3) study of in vitro inhibition of these two pathogens demonstrated specificity of chemical inhibition. He found sodium tetraborate to be relatively more toxic to P. digitatum and the bicarbonate ion more toxic to P. italicum. Van der Plank and Rattray (5) found o-phenylphenol more inhibitory to P. digitatum than to P. italicum. The present in vitro tests showed inhibitory concentrations of 500 to 540 ppm. of C.P. 2229 and 15 to 20 ppm. of Dowicide for P. digitatum. For P. italicum the inhibition points were 550 to 580 ppm. of C.P. 2229 and 70 to 80 ppm. of Dowicide. Thus, the in vitro studies showed that the C.P. 2229 was only slightly specific in its action on the two species of Penicillium. As indicated earlier, the in vivo studies showed little specificity of Dowicide, but its specificity in vitro was marked (Table 2).

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AGRICULTURAL MARKETING SERVICE, BIOLOGICAL SCIENCES BRANCH, QUALITY MAINTENANCE AND IMPROVEMENT SECTION, ORLANDO, FLORIDA.

MANGO ANTHRACNOSE CONTROL IN HAWAII[1]

Minoru Aragaki and Shosuke Goto[2]

Anthracnose of mango (Mangifera indica L.) caused by Colletotrichum gloeosporioides Penz., is very common and widespread in the Territory of Hawaii and is believed to be one of the principal reasons for the unfruitfulness of mango trees in high rainfall areas in Hawaii.

The disease manifests itself in various ways including blossom blight, peduncle blight, leaf spot, fruit stain, and fruit rot (Fig. 1). The blossom blight and accompanying peduncle blight are by far the most destructive phases of this disease. If frequent rainfalls occur during the blossoming period, anthracnose can reduce the fruit set and subsequent yield considerably.

During 1955, Pirie, a mango variety noted to be highly susceptible to anthracnose by Nakasone et al[3]. was used as the test variety for fungicide screening. The materials tested included captan, zineb, ziram, and basic copper sulfate. The first flower buds appeared during the middle of November 1955. Panicles from three trees were tagged so that the course of fruiting could be followed. Spray applications were started on December 1, in the midst of the rainy season, and were continued at weekly intervals during the flowering period and at biweekly intervals thereafter. A total of nine applications was made. Fruit counts were made on January 24, 1956, when the fruits were about half mature. The results are summarized in Table 1.

Table 1. Summary of fruit set counts on January 24, 1956.

Treatment	Rate per 100 gallons in pounds	Number of fruit set*	Number of tagged panicles**	Fruit per tagged panicle
Captan	3	74	100	0.74
Zineb	2	19	90	0.21
Ziram	2	7	80	0.09
Basic copper sulfate	4	2	80	0.02
Check	--	0	70	0.00

* Fruit approximately half mature
** Total from three trees.

Captan gave excellent results in controlling anthracnose while zineb was significantly better than the unsprayed check at the 5 percent level. Captan was significantly better than zineb at the 5 percent level, and better than the rest of the treatments at the 1 percent level.

[1] Published with the approval of the Director of the Hawaii Agricultural Experiment Station as Miscellaneous Paper No. 96.

[2] Junior and Assistant Plant Pathologists, respectively, University of Hawaii, Honolulu, Territory of Hawaii.

[3] Nakasone, H. Y., F. A. I. Bowers, and J. H. Beaumont. 1955. Terminal growth and flowering behavior of the Pirie mango (Mangifera indica L.) in Hawaii. Proc. Am. Soc. Hort. Sci. 66:183-191.

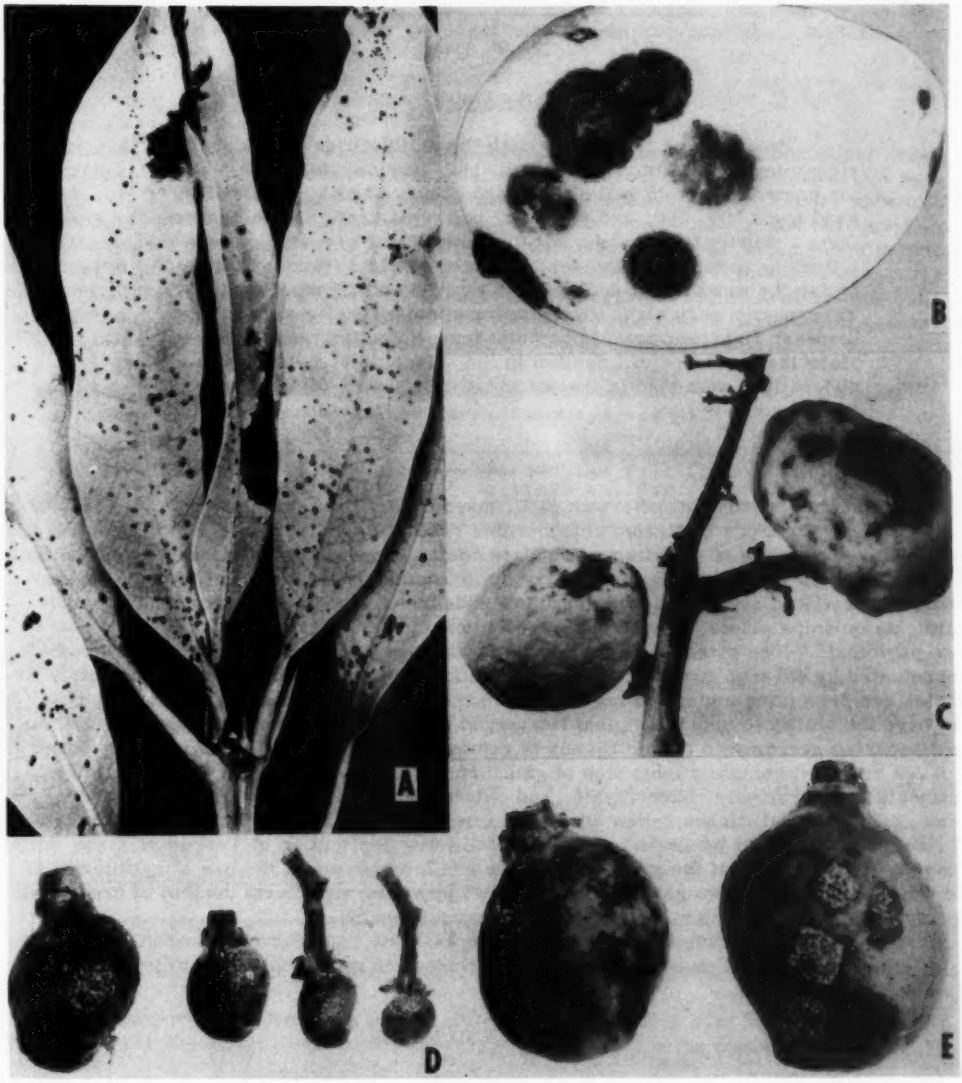

FIGURE 1. Symptoms of Mango anthracnose. A -- on leaves. B -- on mature fruit. C -- on young fruit and peduncle. D, E -- on young fruit at several stages of development.

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REDUCTION IN INCIDENCE OF CROWN GALL OF MAZZARD CHERRY FOLLOWING ANTIBIOTIC TREATMENTS[1]

Ira W. Deep

Summary

Pre-planting treatments with the antibiotic preparations, streptomycin sulfate, Agri-mycin, and Terramycin, were tested for control of crown gall of Mazzard cherry seedlings. Dosages used were 100, 200, and 400 ppm, and treatment periods ranged from 1 minute to 3 hours. All three antibiotic preparations significantly reduced the incidence of infection, but Terramycin was by far the most effective. Certain non-phytotoxic treatments with Terramycin were significantly more effective than a standard Semesan Bel treatment. Chemotherapeutic activity was indicated since the incidence of infection in the uninoculated control was significantly higher than in trees treated in 400 ppm of Agri-mycin for 1 hour.

The economic importance of crown gall caused by Agrobacterium tumefaciens (E. F. Sm. & Towns.) Conn has motivated considerable research to develop a practical control program. The only means of effecting a complete control would be by disinfestation of the soil, but attempts to do this have failed. A partial solution has been treatment of propagation wounds to prevent infection. Mercurial compounds are currently recommended for such treatments on some plants. These are effective surface disinfestants, but they are sometimes phytotoxic. The potential usefulness of antibiotics in controlling crown gall has been demonstrated by several investigators through laboratory and greenhouse experiments. Antibiotics are promising materials because of their bactericidal value and because some of them have the ability to penetrate plant tissue and provide chemotherapeutic action. Blanchard reported aureomycin chemotherapy of crown gall in tomatoes (2). Klemmer, Riker, and Allen reported complete inhibition of gall formation on tomato and marigold following treatments in Terramycin, aureomycin, and chloramphenicol. The action was apparently directed against the gall tissue rather than the bacterium (5). Breakdown of geranium crown gall tissue was reported by Janke and Granits following application of Terramycin and aureomycin to the surface of the galls (4). There are few reports of the use of antibiotics in field trials to control crown gall. Ark and Sibray reported significant control of crown gall of roses by treatments with streptomycin and tetracycline (1).

During this investigation three antibiotic preparations, streptomycin sulfate, Agri-mycin, and Terramycin[2], were tested for effectiveness in root treatments to prevent crown gall (3).

MATERIALS AND METHODS

Mazzard cherry seedling trees, Prunus avium L., were root-pruned, inoculated with a suspension of the crown gall bacterium and allowed to dry for 1 hour. The bacterial culture used had been isolated from Mazzard cherry. The roots were then immersed in an antibiotic suspension for a specified length of time, and the 100 trees used for each treatment were divided into four lots of 25 and planted in randomized blocks. After 6 months the trees were dug and examined for galls.

In 1954 treatments were made with streptomycin sulfate and Agri-mycin (15% streptomycin and 1.5% oxytetracycline) at dosage levels of 100, 200, and 400 ppm for periods of 1, 15, and 60 minutes[3]. Treatments were made with Terramycin (oxytetracycline) for 1 minute at 100, 200, and 400 ppm. All antibiotics used in this investigation were furnished by Chas.

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[1] Technical Paper No. 1111, Oregon Agricultural Experiment Station.
[2] The hydrochloride form of Terramycin at 90% active ingredient was used.
[3] The ppm of each antibiotic refers to total active ingredient.

Pfizer and Co., Inc.

In 1955 the trees were treated for 15 minutes, 1 hour, 2 hours, and 3 hours, in 100, 200, and 400 ppm of streptomycin sulfate, Agri-mycin, and Terramycin. Additional treatments were made with Terramycin at 100, 200, and 400 ppm for 1 minute.

1954 RESULTS

The most significant general trend was consistent decrease in incidence of gall with each increase in length of treatment (Table 1). The treatment period was important as a dosage level. The most effective treatment, 400 ppm of Agri-mycin for 1 hour, appeared to be more effective than the Semesan Bel treatment, but the difference was not significant. The percentage infection following this Agri-mycin treatment, 5.1 percent, was significantly below that of the uninoculated control, 20.2 percent, at the 5 percent level. The uninoculated control was used to determine the percentage of trees with incipient infections but showing no symptoms at the time of treatment (6). The reduction in incidence of gall below the level of incipient infection in the uninoculated control indicates chemotherapeutic activity.

Table 1. The effect of dosage and duration of treatment with three antibiotics on incidence of crown gall of Mazzard cherry trees (1954 Experiment).

Chemical	Dosage	Percentage gall following a treatment for		
	PPM	1 min.	15 min.	1 hour
Streptomycin sulfate	100	26.6	21.5	16.6[a]
	200	26.1	23.9	20.4
	400	25.0	13.6[1]	8.9[a]
Agri-mycin	100	23.3	19.7	13.4[a]
	200	35.5	21.7	11.3[a]
	400	32.2	26.6	5.1[a,b]
Terramycin	100	22.0		
	200	34.8		
	400	18.7		
Semesan Bel	1928	11.9[1]		
Inoculated control		32.5		
Uninoculated control		20.2		

[a] Significantly different from the inoculated control at the 5 percent level.

[b] Significantly different from the uninoculated control at the 5 percent level.

Treatments with streptomycin sulfate and Agri-mycin at 400 ppm for 1 hour appeared to be somewhat phytotoxic, but they did not significantly reduce the stands.

1955 RESULTS

Terramycin was much more effective than either Agri-mycin or streptomycin sulfate (Table 2). The latter two antibiotic preparations significantly reduced infection when used at 200 or 400 ppm for 1, 2, or 3 hours, but treatments at 400 ppm for longer than 1 hour were phytotoxic. Agri-mycin was more effective than streptomycin sulfate; the 400 ppm treatment with Agri-mycin for 1 hour was more effective than the Semesan Bel treatment and was not phytotoxic.

The effectiveness of Terramycin is indicated by the consistent decrease in percentage of gall with increase in dosage and in duration of treatment (Table 3). At dosage levels of 200 and 400 ppm all Terramycin treatments, except that for 1 minute at 200 ppm, were significantly better than the Semesan Bel treatment, but some of these were phytotoxic (Table 4). Treatments at 400 ppm for 1 hour or longer were better than 85 percent effective but were phytotoxic. Treatments at 200 ppm for 2 and 3 hours were also quite effective and were less phytotoxic. The best Terramycin applications with regard to both effectiveness and lack

of phytotoxicity were at 200 ppm for 1 hour or 400 ppm for 15 minutes. The percentage infection following these two treatments was 41.0 and 49.5, respectively, compared to 99 percent in the inoculated control.

Table 2. A comparison of the effectiveness of pre-planting treatments with streptomycin sulfate, Agri-mycin, and Terramycin in preventing crown gall (1955 Experiment).

Treatment		Total percentage gall following treatment with		
PPM	Dosage	Streptomycin	Agri-mycin	Terramycin
100	1 min.	----	----	94.9
	15 min.	94.3	98.9	81.3
	1 hour	95.4	94.4	77.9
	2 hours	80.7	95.7	68.3
	3 hours	89.3	92.9	68.2
200	1 min.	----	----	92.7
	15 min.	97.8	94.7	66.3
	1 hour	93.5	90.7	41.0
	2 hours	74.0	93.2	33.8
	3 hours	86.6	85.6	25.0
400	1 min.	----	----	68.8
	15 min.	82.8	89.5	49.5
	1 hour	82.2	72.9	10.3
	2 hours	85.5	76.6	12.7
	3 hours	62.3	54.2	14.9
Inoculated control		99.0		
Uninoculated control		9.2		

L.S.D. 5% level 3.36
L.S.D. 1% level 5.86

Table 3. The effect of Terramycin treatments on the incidence of crown gall of Mazzard cherry trees (1955 Experiment).

Chemical	Dosage	Percentage gall following treatment for				
	PPM	1 min.	15 min.	1 hr.	2 hr.	3 hr.
Terramycin	100	94.9	81.3	77.9	68.3	68.2
	200	92.7	66.3	41.0	33.8	25.0
	400	68.8	49.5	10.3	12.7	14.9
Semesan Bel	1928	80.2				
Inoculated control		99.0				
Uninoculated control		9.2				

L.S.D. 5% level 3.36
L.S.D. 1% level 5.86

Table 4. The effect of Terramycin treatments on stand of Mazzard cherry trees (1955 Experiment).

Chemical	Dosage	Total stand from 100 trees treated				
	PPM	1 min.	15 min.	1 hr.	2 hr.	3 hr.
Terramycin	100	99	91	87	82	89
	200	96	89	85	80	72
	400	96	93	76	63	72
Semesan Bel	1928	87				
Inoculated control		98 and 91				
Uninoculated control		99 and 89				

L.S.D. 5% level 3.13
L.S.D. 1% level 4.13

The 40 to 50 percent infection that followed these treatments may tend to mask their effectiveness because the inoculum potential was extremely high. Ninety-nine percent of the inoculated control plants were infected, and 80 percent of the plants given the standard treatment with Semesan Bel were infected. It is interesting to compare the Agri-mycin treatment at 400 ppm for 1 hour made in 1954 with the same treatment made in 1955. In 1954 the rate of infection following treatment was 5.1 percent as compared with 32.5 percent in the control. In 1955 there was 72.9 percent infection following the treatment but 99.0 percent infection in the control.

The 1955 experiment gave no evidence concerning the chemotherapeutic activity of the antibiotics. The incidence of incipient infection was lower than in 1954, and the inoculum potential was so high that none of the treatments resulted in complete surface disinfestation. It was therefore not possible to determine whether any internal action occurred.

DISCUSSION

Treatments in Terramycin at 400 ppm for 15 minutes or 200 ppm for 1 hour were much more effective in preventing crown gall of Mazzard cherry than a standard Semesan Bel treatment, and they were not phytotoxic. Cooperative experiments with Oregon nurserymen are now in progress to determine whether these treatments will be beneficial, yet safe under conditions imposed by the growers.

In one trial a treatment with 400 ppm of Agri-mycin for 1 hour eliminated some of the incipient infections. Subsequent greenhouse tests have demonstrated a similar activity of Terramycin. Elimination of incipient infections is an important consideration in controlling crown gall of Mazzard cherry since such infections on seedlings used for lining-out stock may lead to severe losses in nursery stock budded or grafted on the seedlings. During these trials incipient infections have ranged from 6 to 26 percent.

Oregon nurserymen have frequently reported losses in excess of $1000 per acre from crown gall. The use of Terramycin is not a complete control, but these data indicate that crown gall can be greatly reduced by treating roots with Terramycin.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON

PERTINENT ASPECTS OF THE INFECTION CYCLE AND CONTROL OF THE PEACH CANKER PATHOGEN, FUSICOCCUM AMYGDALI[1]

Emil F. Guba

Summary

The infection of the nodes of the herbaceous growth of the peach tree by conidiospores of Fusicoccum amygdali begins in the late summer and early autumn and continues until cold weather. Infection is restricted to the buds. The beginning of the infection cycle coincides with the enlargement of the axillary fruit buds and defoliation in September or shortly after Golden Jubilee peach harvest. The pathogen grows in the buds and thence into the bark about the nodes, constricting and killing the growth above. Pycnidia and conidia of the pathogen appear in great abundance in the cankers from the beginning. These autumn season infections produce a progressive increase in cankers throughout the autumn, winter and early spring, reaching a high ratio of number of cankers to length of shoots with the advent of the spring season. The axillary herbaceous growth, blossoms and young fruits succumb as the nodes of the previous season's shoots are girdled by fungus. The buds may be killed before any new growth takes place.

In May and June another major infection of the buds and nodes of the previous season's growth takes place, terminating when the new herbaceous shoots average about 10 to 12 inches long. Infection of the buds in spring may not cause death of the shoots or constriction of the woody shoot and loss of the fruit until the fruit harvest season. Girdling of the bases and death of the current season's shoots may not be complete until autumn.

INFECTION CYCLE

Infection of peach buds and of the bases of the green axillary shoots by the peach canker pathogen, Fusicoccum amygdali Delacr., beginning at the end of the dormant period in the spring season, was described by Cohoon and Daines (1). These early spring symptoms can also be the result of infection of the buds in the previous autumn (Figures 1-4). Thus the incubation period for infection initiated in the autumn season ranges from 1 to 4 months. Grosclaude (2) reported an incubation period of 2 to 3 months in the orchard. Cohoon and Daines (1) reported that symptoms of cankers in the spring season appear in 1 to 3 months. The factor governing the appearance of the disease is essentially temperature. In southeastern Massachusetts the first nodal cankers appeared on herbaceous shoots on October 3 to 5, 1953, October 5, 1954, October 18, 1955, October 20, 1956, and October 10, 1957. Cohoon and Daines (1) found the first nodal cankers on herbaceous peach shoots in southern New Jersey in August 1953 and 1954 and in September 1955.

The new herbaceous shoots, except for these basal infections, are resistant until the following September. In the event of injury to the cortex of the shoots, caused by hail, insects, or otherwise, with rain added, an epidemic infection can follow in the summer growing season. The cankers appear in 1 month.

The author's tabulations of Fusicoccum cankers began in the autumn season of 1954 and continued each year thereafter (Tables 1, 2). Peach shoots measuring from 6 inches to 5 feet long were collected at random. The prevalence of the disease in the progressive tabulations is based on the number of cankers per 100 linear feet of shoots. It was expected that shoots collected in autumn following a succession of rain infection periods would show more cankers on the second tabulation of the same shoots and an increase in the number of infected shoots. Also it was expected that peach shoots collected in winter after successive rain infection periods in autumn, and held at 70° to 80°F, would show a higher canker count on the second tabulation and more infected shoots. The ratio of cankers to linear feet after incubation at 70° to 80° should be comparable to the ratio appearing in the trees at the end of the winter

[1] Contribution No. 1145, Massachusetts Agricultural Experiment Station.

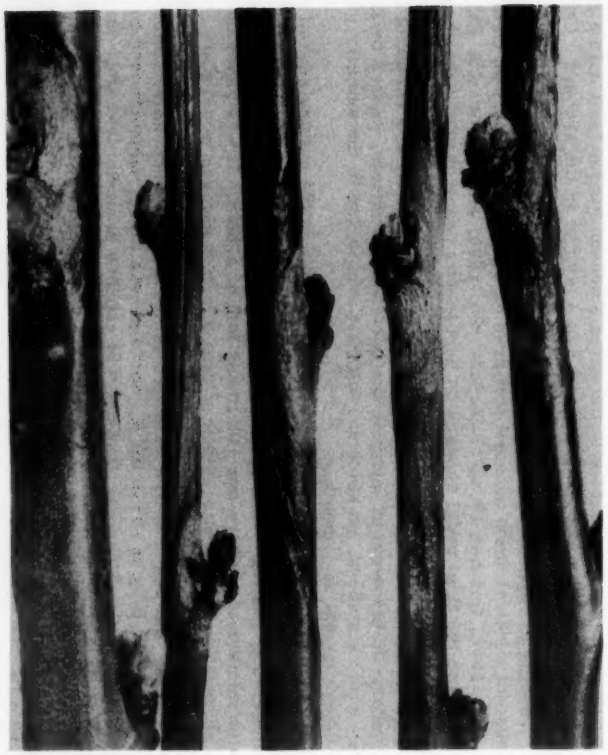

FIGURE 1. Fusicoccum peach cankers arising from infections of the enlarging axillary fruit buds of the herbaceous shoots in the autumn season, December 1952.

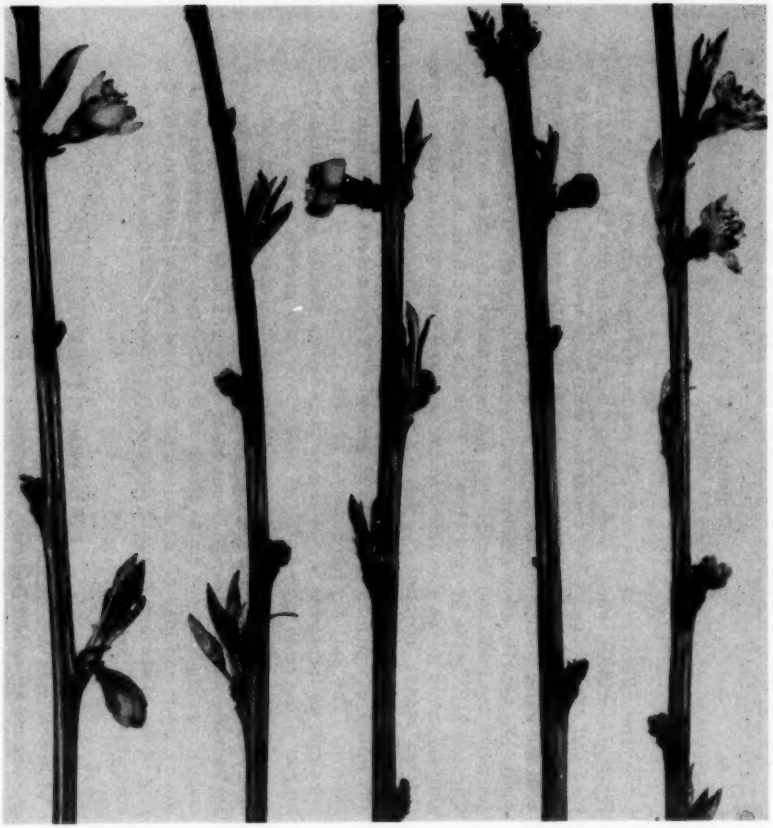

FIGURE 2. Epidemic outbreak of Fusicoccum peach cankers about the nodes (1953 terminal growth) before the blossoming period, April 21, 1954, resulting from conidiospore infections of the buds in late autumn 1953.

FIGURE 3. Woody shoots of 1952 and axillary herbaceous shoots of 1953 growth (July 15, 1953) showing Fusicoccum peach cankers about the nodes resulting from infection of the buds and nodes in late autumn 1952 and spring 1953. The woody and herbaceous growth above the cankers is destroyed.

FIGURE 4. Woody peach shoots, 1951 season, and axillary woody shoots, 1952 season, in December 1952, showing disease resulting from infections in the autumn 1951 and spring 1952 seasons combined. The Fusicoccum cankers are invaded by secondary pathogens from the beginning, notably by Cytospora (Valsa leucostoma Pers. ex Fr.).

Table 1. Number of Fusicoccum cankers to 100 linear feet of peach shoots, variety Golden Jubilee, Wilson Orchard, Raynham, Massachusetts, November 1952 to January 1958.

Date	: Number of cankers on current season's : shoots (100 linear feet)
I. No Autumn Season Fungicide Schedule 1952, 1953, 1954	
November 2, 1952	Cankers abundant
December 10	Cankers epidemic
April 24, 1953	Trees coming into bloom. Abundance of cankers in 1952 shoots.
September 15, 1953	0
October 3	First cankers
November 19	Cankers abundant
April 21, 1954	Trees coming into bloom. Abundance of cankers in 1953 shoots.
September 17 - October 8, 1954	0 to rare
November 17	58.2
November 30	82.0
December 14	94.0
December 27	143.0
January 1, 1955	82.0
January 15	115.0
February 11	85.0
February 27	94.0
March 7	93.0
March 20	114.0
April 1	83.0
April 15	107.0
May 4	95.1
May 12	145.4
II. Autumn Schedule of Fungicide Applications	
IIa. Ferbam 1 1/4 lb. and Phygon 1/4 lb., Aug. 24, Sept. 1, 26, Oct. 2, 10, 25, 1955.	
September 20, 1955	0
October 18	First cankers apparent
November 6	17.0
November 18	20.0
November 26	38.3
December 5	46.6
January 1, 1956	20.6
January 9	49.0
January 24	46.0
February 6	58.7

Table 1. (Continued)

IIa. (Continued)

February 16		30.0
February 27		30.8
March 27		35.6
April 2		35.6
May 2		35.0
May 11		63.0[a]

IIb. Thylate 1 lb. with Glyodin 1 pint, on Sept. 17 and Oct. 1; Ferbam 1 1/4 lb., Phygon 1/4 lb., Glyodin 1 pint, on October 4, 7, 8, 27, 1956.

October 9, 1956		0
October 24		7.0
November 5		19.5
	no fungicide	87.0
November 6		16.6
November 19		33.3
November 23		30.2[b]
December 3		35.2[b]
December 17		25.6
December 26		33.2
March 6, 1957		25.3
March 14		25.3
	no fungicide	171.0
April 22		23.6

IIc. Thylate 1 1/2 lb., Phygon 1/4 lb., Glyodin 3/4 pint on Sept. 9, 18, 30; October 10, 24; November 4: Commercial Liquid Lime Sulfur 6 1/2 gallons, Glyodin 3/4 pint, on November 14 when 90% of the leaves were shed, 1957.

October 16, 1957	0.0
October 26	0.0
October 18	2.27
October 28	2.27
November 2	6.84
November 11	7.82
November 14	12.6
November 26	13.6
December 4	5.6
December 16	9.1
December 27	5.8
January 10, 1958	7.54

[a] Brown Rot epidemic

[b] Of the total number of cankers in these tabulations 14.2% are restricted to the terminal bud.

Table 2. Number of Fusicoccum cankers to 100 linear feet of peach shoots, variety Golden Jubilee, Leonard Orchard, Raynham, and Travares Orchard, South Lakeville, January 1953 to January 1958 inclusive.

Date	Number of cankers per 100 linear feet		
	Leonard		Traverse
January 1, 1955	31	60	27
January 19	57	96*	31
March 7		29	36
March 15		46	52
May 4	3	6	
May 12	3	34	
November 6		4	57
November 18		7	68
May 28, 1956		64	
October 24		5	42
November 5		7	87
March 6, 1957		22	91
October 16, 1957	0	7.68	
October 26	0	7.68	
October 18	1.35	0.0	
October 28	2.70	5.85	
November 2	0.0	12.54	
November 11	0.0	12.54	
November 14		0.0	
November 26		2.4	
December 4		2.3	13.02
December 16		2.3	19.53

* Leonard orchard well pruned after January 1, 1955 and before next canker count March 7, 1955.

season. Generally these expectations proved to be true.

The first cankers on the green herbaceous shoots appear in October in southeastern Massachusetts. The incubation period is 1 month at this time, thus the autumn infection period begins in rain periods in September. This is after the Golden Jubilee harvest season, which is normally August 24 to September 1 in southeastern Massachusetts. A study of tabulations of cankers, monthly rainfall, and mean temperatures over a period of years, discloses the correlation between heavy rainfall and epidemic outbreaks of cankers; also the influence of temperature upon the incubation period of the disease. All of the infections may express symptoms before the end of the autumn or winter seasons. In the event of heavy rainfall late in autumn and early onset of cold weather, the progression of new cankers will end with an epidemic outbreak just prior to the blossoming period in the following spring (Table 3).

The golden Jubilee variety showed a mild incidence of new cankers in autumn of 1951, 1952, and 1957, moderate in 1953 and 1956, and serious in 1954 and 1955. Development of cankers was mild after the winter of 1952-1953, moderate after 1956-1957, and serious after 1951-1952, 1953-1954, 1954-1955, and 1955-1956. In February 1954 an abundance of dead

fruit buds was noted at Raynham, and in March an epidemic outbreak of cankers followed as a sequence to the decay of the buds. There was an epidemic occurrence of cankers in the mild winter of 1954-1955. The spring seasons of 1953 and 1954, particularly, were marked by heavy rain infection periods and were followed by an epidemic of dead leaf clusters and flagging shoots. Thus the variation in the prevalence of cankers in the autumn and spring seasons closely follows the amount of rainfall in these periods (Table 3).

The tabulations also reflect the effect of the growers' control program, as well as variations in amount of disease due to conditions peculiar to each orchard (Table 2).

Cohoon and Daines (1), in southern New Jersey, reported a progressive increase in new cankers from August to November 1953 inclusive, beginning with none in July. The increase was consistent but relatively less in December 1953 to March 1954 inclusive, being least in January, and was greatest in April, June, and May 1954, respectively. In the period 1954 to 1955, the autumn season increase was consistently progressive. The greatest increase occurred in October 1954. It was relatively less from November 1954 to March 1955. The greatest increase of new cankers in the 1954-1955 season appeared in April to June, 1955 inclusive. The period of greatest increase noted in their investigation, which was conducted from August 1953 through January 1956, occurred in October to November inclusive, 1955, and followed the highest rainfall recorded during their entire study. The small increases in new cankers in the winter seasons, December 1953 to March 1954, November 1954 to March 1955, and December 1955 to January 1956, reflect the influence of low winter temperatures upon the incubation of the disease.

TEMPERATURE ASPECTS OF INOCULATION AND INFECTION

Dormant peach shoots bearing cankers were set upright in shallow water in covered glass jars and placed in constant temperature chambers registering 40°, 45°, and 50° F. At 50°, tendrils of conidia issued from the pycnidia after the third day (72 hours), becoming longer and more abundant on each successive day. At 45° exudation of conidia began on the sixth day, and the number of exuding pycnidia increased with further incubation. On the thirteenth day, exudation was general, but the conidial cirrhi were mostly very short. At 40°, after 7 days no cirrhi were observed; on the fourteenth day some pycnidia were crowned with a miniature cone of conidia. Thus the studies show that temperature plays an important part in the exudation of the conidia.

Conidiospores of Fusicoccum amygdali failed to germinate in 14 days at 40°F (end of test). At 45° there was no germination on the fourth day; on the sixth day 2 to 3 percent of the conidia were germinated with germ tubes averaging 6.2 microns long. At 50°, germination appeared on the fourth day; on the sixth day 80 to 90 percent of the conidia were germinated, with germ tubes averaging 24 microns or about 3 1/2 times the length of the conidia. In the coastal area of southern New England, the monthly mean maximum temperature is below 50° from late November to March inclusive. It is below 45° from December to mid-March. The monthly mean minimum temperature is below 50° from October through May, bordering on or below 40° from November through April, and below 30° from December through early March. Exudation and germination of conidia in the dormant winter season would require at least 7 successive days of a mean daily temperature of 50° with moisture, and additional time for infection. This condition would hardly prevail in southeastern Massachusetts.

The growth-temperature curve of the pathogen shows a very slow rate of growth at 40° to 50°F (Figure 5). Occurrence of such temperatures and even higher ones for brief periods during the winter season explains the appearance of new cankers from incipient infections and the increase in the importance of the disease, but not the development of new cankers from fresh conidial infections. Thus, successful artificial infection of dormant buds in the orchard in early winter with atomized conidia in water would appear to be inconsistent with natural infection phenomena, since temperature controls the swelling of the pycnidia and eruption of conidia, as well as germination of the conidia and infection. In southeastern Massachusetts, the exudation of conidial tendrils is not resumed until late April and May. The first foliage infections of the season appear in middle to late June. The author has never observed tendrils of conidia issuing from pycnidia in the dormant season, even during periods of mild weather. It would appear that the same circumstances apply farther south in the Atlantic coastal area, with a similar climate. For example, in southern New Jersey, the monthly mean maximum temperature is below or about 50°F from late November to the latter part of March. The monthly mean minimum temperature is below or about 50° from October to May inclusive. The months of December to February inclusive are coldest, with the mean minimum temperature below 30° and the monthly mean maximum temperature below 50°.

Table 3. Monthly Rainfall, inches, Taunton (Raynham), Massachusetts August to December and April to June inclusive, 1951-1957.

		AUG.	SEPT.	OCT.	NOV.	DEC.		APR.	MAY	JUNE
	1951	3.36	1.32	2.65	7.28	3.71	1952	2.90	3.56	1.98
Departure from Normal	"	-.68	-2.39	-1.14	+3.22	-.22	"	-1.23	+.20	-1.23
	1952	6.63	1.73	1.86	2.46	2.86	1953	4.57	3.64	.32
Departure	"	+2.59	-1.98	-1.93	-1.60	-1.07	"	+.39	+.28	-2.89
	1953	3.53	3.39	5.10	7.99	5.11	1954	4.56	7.86	2.62
Departure	"	-.51	-.32	+1.31	+3.93	+1.18	"	+.43	+4.50	-.59
	1954	7.32	6.61	2.82	2.87	4.69	1955	2.48	1.75	2.69
Departure	"	+3.25	+2.90	-.07	-1.19	+.76	"	-1.65	-1.61	-.52
	1955	15.78	3.33	8.49	5.51	.62	1956	2.74	2.52	3.10
Departure	"	+11.74	-.38	+4.70	+1.45	-3.32	"	-.48	-.84	-.11
	1956	3.32	3.23	4.24	3.49	4.90	1957	4.39	1.50	.50
Departure	"	-.72	-.48	+.45	-.57	+.97	"	+.26	-1.86	-2.71
	1957	2.80	.71	2.33	4.16					
Departure	"	-1.24	-3.00	-1.46	+.10					

Table 4. Percentage of cankers distributed among each quarter length of 1955 growth, Golden Jubilee variety, January 25, 1956.

GROWTH of 1955	Average Length Feet	QUARTER LENGTHS FROM BASE TO TERMINAL BUD			
		0 - 1/4	1/4 - 1/2	1/2 - 3/4	3/4 - 4/4
Terminals	1.07	2.0	6.5	24.5	67.0
Shoots	3.40	0.6	2.6	33.9	62.9

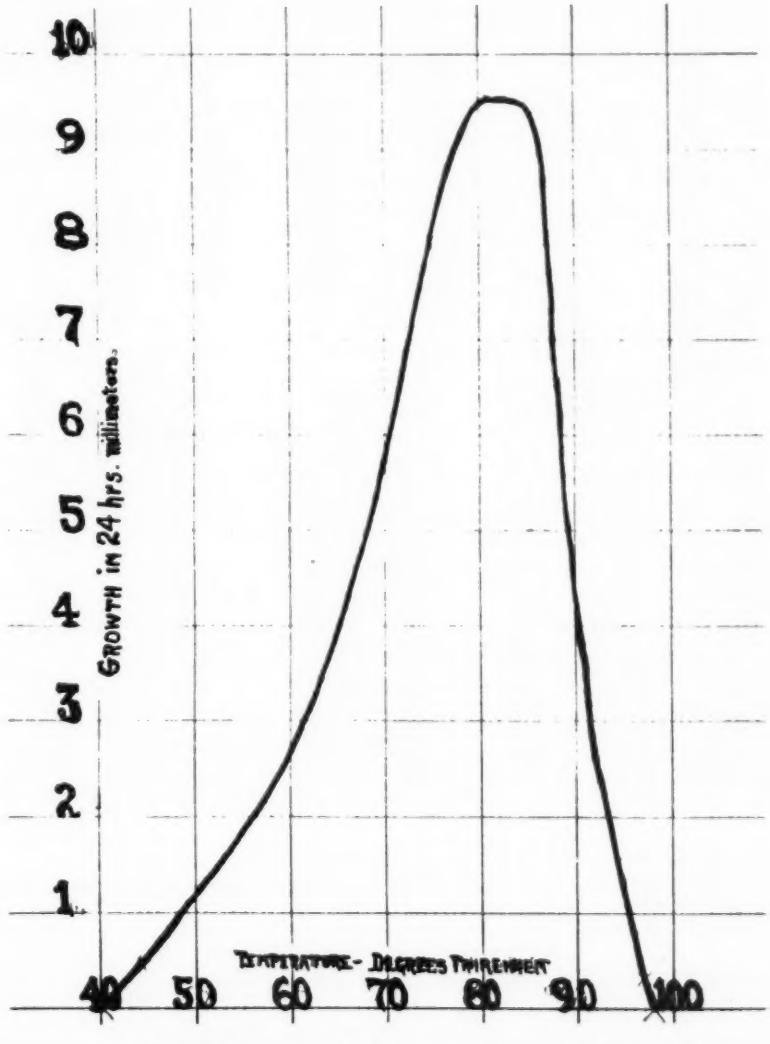

FIGURE 5. Growth-temperature curve of the peach canker fungus Fusicoccum amygdali. Optimum temperatures for growth 80° to 84° F; the extreme temperatures 40° to 98°.

INFECTION COURTS

The author finds that infection is directly into the bud itself and not at the bud scale and leaf scars. It coincides with the swelling of the fruit buds (Figure 6) and is independent of the shedding of the leaves. At first infection invades the whole bud or portions of it, then the bud peduncle and the bark under the scars and around the node. The infection period is terminated by low temperatures in autumn. It is resumed in the spring season with the advent of temperatures favorable for conidial exudation and germination.

FIGURE 6. Beginning of Fusicoccum peach cankers. Vertical sections through buds and nodes showing the progress of infection from infected buds into cortex of the shoots, May 2, 1956.

Fusicoccum is a wound pathogen. Enlargement of the axillary fruit buds is associated with defoliation in autumn. The pubescent bud scales are pushed aside. The apical portions of the scales are brown and dead, and there is considerable dead loose scale debris about the buds.

Infection courts are created only in some of the buds, notably among those of the latest growth of shoots and terminals. Favorable conditions can be the result of heavy rainfall and a high soil water table in the latter part of the growing season. In spite of a tremendous amount of conidial inoculum in the trees, infection is not general among all of the nodes of the latest growth, but selective, as the pattern of infection on water sprouts and terminals shows (Table 4).

Tabulation of cankers on the terminal growth of 1955, on January 25, 1956 (Table 4) showed most cankers above the middle, with the greatest number in the last quarter of the length of the terminals. The last half of the shoot length contained 96.8 percent of the total number of cankers. The greatest number of cankers were distributed among the nodes in the last quarter of the length. In some seasons a high percentage of the terminal buds is infected. Thus the infection courts are in the buds, largely in the latest portion of the herbaceous growth.

Previous studies have represented bud scale and leaf scars as wounds and infection courts. Grosclaude (2) emphasized the importance of wounds to infection, and stated that the fragile buds could be wounded in swelling and opening. He obtained infection in both artificially wounded and unwounded buds. A cover crop in the orchard would appear to act as a deterrent to infection by sharing in water use and thus decreasing the amount of excess moisture for tree growth. An abundance of healthy foliage would also appear desirable indirectly as a deterrent to infection.

CONTROL WITH FUNGICIDES

The prevention of autumn infections is crucial to the control of the Fusicoccum canker disease. These cankers represent the chief source of inoculum for progression of the disease in the following year. A loose schedule of fungicide applications in the autumn season of 1955 (Table 1, IIa) with poor equipment gave some control. The circumstances were somewhat similar in autumn of 1956 (Table 1, IIb). In autumn of 1957, (Table 1, IIc) protection was thorough and more frequent; also the weather was dry. The progressive decline in the prevalence of the disease in the Wilson orchard from 95.1 and 145.4 cankers per 100 linear feet (1954-growth shoots) in May 1955, to 7.54 cankers (1957 shoots), in January 1958, is impressive.

The same program, combined with persistent pruning and sanitation, has almost eliminated the Fusicoccum disease in the Leonard orchard, where the number of cankers in 100 linear feet of herbaceous shoots was 31 and 57 in October 1954, and 60 and 96 in January 1955, whereas in December 1957 it was 3.6 cankers (1957 growth).

The last fungicide application in autumn is made when 90 percent of the leaves have fallen, of liquid lime sulfur concentrate, 7 gallons in 100 gallons of water, and Glyodin, 3/4 pint, to disinfect the buds and to provide lasting protection. The application is repeated at late to delayed dormant in the following spring, followed by the usual spray applications for early brown rot control and by all the summer cover sprays. All applications include 3/4 pint of Glyodin to 100 gallons of diluted spray. The coverage of the buds is unsatisfactory without Glyodin.

The spray schedule recommended for the control of Fusicoccum canker in southeastern Massachusetts is as follows:

Immediately after Fruit Harvest -- Beginning in early September and continuing at 10-day intervals, or in advance of rain periods, until 90 percent of the leaves have fallen (variety Golden Jubilee): Thiram or ferbam or captan 2 pounds, Glyodin 3/4 pint; or, thiram or ferbam 1 1/2 pounds, Glyodin 3/4 pint.

When 90 percent of the Leaves Have Fallen: Lime sulfur concentrate 7 to 10 gallons, Glyodin 3/4 pint.

Late Dormant as for Leaf Curl: As above.

Pink, Blossom Shuck Split as for Brown Rot Control: Same as after-harvest sprays. Do not use dichlone in blossom period and after shuck split.

Cover Sprays: Same as after-harvest sprays. Do not use ferbam after first week in July.

The fungicide program is supplemented by thorough and persistent pruning of diseased branches in the dormant season and in the spring growing season, to reduce inoculum to the minimum.

On the basis of the author's work a badly diseased orchard may be rehabilitated in 3 years.

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MASSACHUSETTS AGRICULTURAL EXPERIMENT STATION

THE FUNGICIDAL EFFICACY OF CYPREX AGAINST APPLE SCAB[1]

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Summary

Cyprex (70% n-dodecylguanidine acetate) used at rates of 3/4, 1, and 1 1/2 pounds to 100 gallons of water was equally effective at all concentrations in producing 99 percent control of scab infection as compared with no control on untreated trees. Other materials tested included Thioneb 50W, dichlone 50W, Crag W. P., Omadine (zinc), Panogen, captan 50W, TD 40, Puratized 1180 and 1143, and phaltan. Panogen at 1/2 and 1 pint to 100 gallons of water gave 70 and 82 percent control respectively. The other materials ranged from 45 to 57 percent effective.

Cyprex at the rate of 2 pounds to 100 gallons of water was sprayed on the trees of different plots 3, 4, 5, 6, 7, and 8 times. Foliage infection at harvest on these plots was 88, 71, 52, 44, 28, and 23 percent, respectively, with the untreated trees showing 95 percent infection. In contrast to this degree of foliage infection, the number of fruits infected at harvest varied between treatments from 0 to 1 percent. Cyprex apparently protects the fruit from scab infection over an extremely long period of time, in comparison with other fungicides have been similarily evaluated. The 2-pound dosage protects the foliage satisfactorily for approximately 30 days. Detailed observations revealed that much of the increased foliar infection occurred on leaves that developed after the last application of Cyprex, thus they had never been treated.

The analyses of fruit residues showed that n-dodecylguanidine acetate dissipates rather rapidly under field conditions. Application of the 2-pound concentration increased residual load on the fruit to a maximum of approximately 5 ppm. Heavy rainfall reduced deposits to less than 1 ppm, but the residue never completely disappeared.

INTRODUCTION

From preliminary experiments in 1956[3, 4] it was found that Cyprex (70% n-dodecylguanidine acetate) was highly effective in controlling apple scab. As a result of this discovery the work with Cyprex was expanded in 1957. Tests were devised to compare Cyprex with other fungicides and to evaluate its residual effectiveness in controlling apple scab. Residual toxicity data were obtained by making infection counts at intervals during the summer and by analyses of fruit residues.

METHODS AND RESULTS

Test 1. A Field Comparison of the Fungicidal Efficiency of Cyprex with other Chemicals.

Three-year-old Clark dwarf apple trees of the varieties Jonathan, Golden Delicious, and Starking were selected so that each treatment was replicated three times on each variety. Sprays were applied with a single-nozzle Bean Spraymaster gun with a 6/64-inch aperature at 300 pounds pressure. A flex-board shield 8 feet square attached to the spray machine was

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[3] Powell, Dwight. 1956. The effectiveness of some new fungicides and combinations of fungicides for control of apple scab. Trans. Illinois Hort. Soc. 90: 142-145.

[4] Results of 1956 fungicide tests. American Phytopathological Society Report. Agricultural Chemicals, February-July. 1957.

Table 1. Rainfall for April through July, and maximum temperatures for April and May; Urbana, Illinois, 1957.

Date	Inches of Rainfall				Daily Temperatures (°F.)			
	April	May	June	July	April		May	
					Max.	Min.	Max.	Min.
1	.71	B			55	49	73	54B
2	.01				49	38	69	50
3	1.65			.01	39	32	79	46
4	.16			.05	57	37	84	33
5	.14				47	34	88	59
6			A.03		52	34	88	65
7	.18		.09		46	33	76	61
8	.77		.01		38	31	73	57
9		A.09B			53	31	83	61
10		.18			55	35	85	67
11	.04	.05	.84	B	46	27	84	69
12		.76	.42		38	23	83	65
13	T	.53	1.16	2.76	45	26	79	64
14			A.31		49	28	82	66
15	.03		T		54	33	85	66
16	.27	T		.02	52	42	90	72
17	.14	.93	.03B	.14	61	50	91	73
18	.01	T	.82		73	54	85	63
19	.04	.18		B	79	54	82	56
20	.87		A		74	63	84	59
21		.30			74	50	84	62
22	A[a].18	.09		.71	76	48	87	66
23	.03	.27		1.39	81	62	83	65
24	.07	A B	.03		80	64	78	60
25	A .20	.34			77	62	84	57
26	1.22B[b]				80	60	83	64
27	.58		.59B	T	65	54	81	65
28	.71		2.11		64	54	77	65
29					74	51	85	62
30		A T B	.06	.01	79	49	85	65
31		.36		B				
Total	7.49	4.08	6.46	5.09				

[a] A. Spray dates for miscellaneous fungicide block.

[b] B. Spray dates for Cyprex residue block.

used to minimize drift during the application of the sprays.

In addition to Cyprex, the following materials were used: Thioneb, 50% wettable powder of polyethylene thiuram sulfide; dichlone, 2,3-dichloro-1,4-naphthoquinone, 50% wettable powder; Crag W.P., 75% wettable powder of 2-heptadecyl glyoxalidine-free base; Omadine, 50% wettable powder of zinc salt of 2-pyridinethione 1-oxide; Panogen, 2.2% methylmercury dicyandiamide (1.5% mercury); captan, N-trichloromethylmercapto-4-cyclohexene-1,2-dicarboximide, 50% wettable powder; TD 40, 40% dinitro benzene thiocyanate; phaltan, a tetrahydro analogue of captan; Puratized 1143, a carbamate of quaternary ammonium; and Puratized 1180, a bisdithiocarbamate of quaternary ammonium.

The dates on which the trees were sprayed are as follows: April 22, prepink; April 25, pink; May 9, calyx; May 24, first cover; May 30, second cover; June 6, third cover; June 14, fourth cover; and June 20, fifth cover. Starting with the second cover, lead arsenate was added to all sprays. After the fifth cover the fungicide program was stopped and insecticide

Table 2. A comparison of Cyprex with other fungicides when used for apple scab control.

Treatment	Dosage per 100 gallons	Percent foliage infection		Percent control
		May 27	July 18	July 18
1 Thioneb 50W	1/2 lb.	15	76	15
2 Thioneb 50W	1 lb.	14	73	18
3 Cyprex	3/4 lb.	2	1.3	99
4 Cyprex	1 lb.	2	0.2	99
5 Cyprex	1 1/2 lb.	0.9	0.2	99
6 Phygon	5 oz.	11	74	16
7 Phygon	10 oz.	9	48	46
8 Crag W.P.	1 lb.	12	49	45
9 Omadine	2 lb.	12	54	40
10 Panogen	1/2 pt.	13	27	70
11 Panogen	1 pt.	11	16	82
12 Captan 50W	2 pt.	12	52	41
13 TD 40	2 lb.	12	38	57
14 Puratized 1180	2 lb.	10	43	52
15 Puratized 1143	2 lb.	11	41	54
16 Check	---	32	89	0
17 Phaltan	2 lb.	24	49	45

sprays containing 50% DDT 1 1/2 pound, and 25% malathion 2 pounds, to 100 gallons of water were applied at frequent intervals.

Scab infection data were taken May 27 and July 18 by examining 50 leaves of each tree and recording the number of leaves showing infection. Fruit infection data were not taken because there was not a sufficient number of fruit for an adequate record.

The rainfall records for April, May, June, and July, and minimum and maximum daily temperatures for April and May are given in Table 1. The rainfall from April 22 to 28 created an ideal situation for maximum primary infections. In addition, the total rainfall of 7.49 inches for April made it impossible to work in the orchard with the spray equipment from April 25 through May 9. After the May 9 spray, almost continuous rain until May 24 caused a similar condition. Thus, two additional sprays would normally have been applied during the May 1 and 16 periods had inclement weather not interfered. Spraying on these dates would have increased greatly the effectiveness of the non-residual materials such as dichlone, and in general, tests with all of the chemicals would have been more satisfactory.

Table 2 lists the treatments, and indicates the concentrations and their effectiveness in controlling scab. Cyprex was superior to the other treatments. No significant differences between the 3/4-, 1-, and 1 1/2-pound dosages were noted. These tests indicate that the 3/4-pound concentration would be adequate for control in a commercial orchard. Panogen was second to Cyprex in efficiency even with the 1/2-pint dosage. Puratized 1143, 1180, and TD 40 were superior to captan. Crag W.P., phaltan, and Omadine probably would have shown considerably more promise under near-normal conditions. None of the materials caused noticeable injury to the trees.

Test 2. Residue Studies with Cyprex.

Eight-year-old trees of the varieties Jonathan, Golden Delicious, Rome Beauty, and Duch-

ess were used for this experiment. Sprays were applied with a 28-E Bean Speed sprayer using a dilute concentration of spray materials. The following spray schedule gives the materials used to 100 gallons of water:

March 18, dormant	: Dn 289, 2 quarts
April 28, prepink	: Cyprex 2 pounds
May 1, pink to early bloom	: Cyprex 2 pounds
May 9, calyx	: Cyprex 2 pounds, lead arsenate 2 pounds
May 24, first cover	: Same as calyx
May 30, second cover	: Same as calyx
June 17, third cover	: Same as calyx
June 27, fourth cover	: Cyprex 2 pounds, 50% DDT 1 1/2 pound, 25% malathion 1 1/2 pound
July 11, fifth cover	: Same as fourth cover
July 17, sixth cover	: 50% DDT 1 1/2 pound, 25% malathion 1 1/2 pound
July 30, seventh cover	: Same as sixth cover
August 6, eighth cover	: Same as sixth cover

All trees except the untreated check received the first three foliar applications. After the calyx spray certain trees of each variety were selected and tagged. The tagged trees were sprayed only with an insecticide during the remainder of the season. This same procedure was followed for the remaining cover sprays so that there were finally seven different plots as follows: Check plot, no fungicide spray; Plot 1, three Cyprex sprays; Plot 2, four Cyprex sprays; Plot 3, five Cyprex sprays; Plot 4, six Cyprex sprays; Plot 5, seven Cyprex sprays; Plot 6, eight Cyprex sprays. Except for Duchess all varieties were treated equally. Duchess is an early summer variety, thus it was not treated after June 27.

Fruit samples were collected for residue analyses from Duchess June 19, 25, July 3, and at the harvest date of July 30. Similar samples were collected from Jonathan June 19, 25, July 3, 10, 11, 14, August 6, and at harvest on September 3. Harvest samples were taken of Golden Delicious and Rome Beauty on October 1. Fruits were selected at random at eye level around the periphery of the tree. Ten or more fruits were selected from each of three trees per treatment for each analysis, placed in previously weighed glass jars, and stored at 33°F until analyzed. The method of analysis used was that outlined by the American Cyanamid Laboratories, Stamford, Connecticut.

The percentage of scab infection on both the foliage and fruit after treating with Cyprex is given in Tables 3 and 4. Since there was a continuous source of inoculum from unsprayed trees adjacent to the treated trees throughout the experiment, one could expect tremendous increases in the amount of infection on trees not properly protected. The last fungicide spray on Plot 1 was on May 9, therefore, it is significant that scab had not developed markedly by June 19. Plot 2, which was sprayed last on May 24, showed an increase in scab infection at the July 16 count but still showed less than Plot 1. Plot 3 was sprayed last on May 30, but still showed only 2 percent foliage infection by July 16. These data indicate that Cyprex gave adequate protection for approximately 30 days. On the basis of these results, in a commercially operated orchard where the entire orchard would have been treated leaving no source of secondary infection, five sprays of Cyprex, 2 pounds to 100 gallons of water, would be sufficient for foliar protection for the entire season.

It is shown in Table 4 that almost perfect control of scab infection on the fruit was secured by the three early sprays. This is important in the evaluation of Cyprex, since both Jonathan and Rome Beauty fruits are highly susceptible to scab. The fruit apparently retains a sufficient amount of the toxicant on the surface to prevent infection for a long period of time.

The residue data are shown in Tables 5 and 6, for Duchess and Jonathan varieties, respectively. Except as influenced by rainfall, Cyprex appears to be a fairly stable material. Analysis of fruits from the Jonathan Plot 6 on July 10 (Table 6) showed a deposit of n-dodecylguanidine acetate of 2.8 ppm. After the spray of July 11 the deposit had increased to 7.8 ppm. Following a rain of 2.76 inches on July 13 the deposit was reduced to 1.4 ppm, a loss of 82 percent. The data would indicate that once the initial loss of residue occurred, there was a gradual decrease over a comparatively long period of time. The continuous increase in fruit size would cause a gradual loss, and this, plus the influence of weather, may be responsible for the decrease of residual deposits. The maximum increase of n-dodecylguanidine acetate deposit to occur from any one application was 5.1 ppm. The highest accu-

Table 3. The percentage of apple leaves of varieties Jonathan, etc., infected with scab after spraying with Cyprex 2 pounds to 100 gallons, as compared with no treatment.

Plot no.	Number of sprays	Percent leaves infected		
		June 18	July 16	September 24
1	3	4	30	88
2	4	3	16	71
3	5	1	2	52
4	6	1	1	44
5	7	--	1	28
6	8	--	0.1	23
Check	None	20	60.2	95

Table 4. Percentage of apple fruit of varieties Jonathan, Golden Delicious, Rome Beauty and Duchess, infected with scab after treatment with Cyprex 2 pounds to 100 gallons, as compared with no treatment.

Plot no.	Number of sprays	Percent fruit infected		
		June 25	July 10	September 23[a]
1	3	0.9	0	0
2	4	0.4	0.9	0
3	5	1.0	0	0
4	6	0.4	0	1.0
5	7	0.2	0	0.8
6	8	--	0	0
Check	0	36.2	32.9	50.4

[a]This count does not include the Duchess variety.

Table 5. Residue at indicated sampling dates from sprays applied at dates shown (x), of n-dodecylguanidine in ppm and average in grams, per apple (variety Duchess).

Date	Residue at indicated dates						
	Ppm per fruit						Average in grams per fruit
	Plot 1	Plot 2	Plot 3	Plot 4	Plot 5	Check plot	
April 25	x	x	x	x	x		
May 1	x	x	x	x	x		
May 9	x	x	x	x	x		
May 24		x	x	x	x		
May 30			x	x	x		
June 17				x	x		
June 19	1.08	.76	1.23	3.15	3.15	.51	31.6
June 25				2.95	2.95		42.5
June 27					x		
July 3					2.76		59.7
July 30	.06	.08	.09	.36	.65	.03	114.0

Table 6. Residue at indicated sampling dates from sprays applied at dates shown (x), of n-dodecylguanidine in ppm and average in grams, per apple (variety Jonathan).

	Residue at indicated dates							
	Ppm per fruit							Average
	Plot 1	Plot 2	Plot 3	Plot 4	Plot 5	Plot 6	Check plot	in grams per fruit
April 25	x	x	x	x	x	x		
May 1	x	x	x	x	x	x		
May 9	x	x	x	x	x	x		
May 24		x	x	x	x	x		
May 30			x	x	x	x		
June 17				x	x	x		
June 19	.13	1.30	1.57	5.69	5.69	5.69	0	15.6
June 25				2.89	2.89	2.89		18.9
June 27					x	x		
July 3					4.27	4.27		26.0
July 10					2.80	2.80		35.5
July 11						7.90 x	.60	34.8
July 14						1.40		41.8
August 6	.09	.10	.23	.26	.19	.33	.09	72.5
September 3	.03	.19	.16	.17	.26	.49	.03	93.56

mulation to occur after any one spray was 7.9 ppm on the Jonathan after the eighth application (Table 6). The harvest residue data for Jonathan and Duchess show that none of the plots exceeded 0.49 ppm. The residues at harvest on Golden Delicious and Rome Beauty varied between 0.01 and 0.14 ppm from the different plots.

DISCUSSION

Cyprex was superior to any other material tested for the control of apple scab. It would appear, from the experimental results reported, that when used commercially the dosage need not exceed 3/4 pound to 100 gallons. Since no phytotoxicity has been observed in the two years that Cyprex has been tested on apple at the Illinois Experiment Station, even at the higher dosage of 2 pounds, the 3/4 pound concentration should be safe to use.

More work needs to be done on the mode of action of Cyprex. Thus far, it has demonstrated highly fungicidal residual effects over long periods of time. There is also some indication that it may perform as an eradicant. The analyses of residue deposits show that Cyprex does not build up excessively. It is easily removed by rainfall. With none of the harvest residues exceeding 0.49 ppm after as many as eight applications of the 2-pound dosage, it would appear that residues will not be a problem in the harvested fruit. Evidence that the 3/4-pound dosage may be sufficient indicates that the problem of objectionable residue on the fruit at harvest may be further minimized in future use of Cyprex.

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CONTROL OF BULL'S EYE ROT OF APPLE IN NORTH CENTRAL WASHINGTON[1]

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Of the several organisms that cause a target spot or bull's eye type of apple fruit decay, the anthracnose fungus, Gloeosporium perennans Zeller and Childs, is usually the most common in North Central Washington. Because this fungus can infect the fruit from petal fall until harvest its control is difficult. Although infection may occur early, actual evidence of its presence develops only when the fruit ripens at the end of cold storage or soon thereafter.

The recommendations for control of bull's eye rot are an early spray of ziram or captan at or just after petal fall, again in late August, and, in the case of late varieties such as Winesap, a third spray in mid-late September. Often the third spray on late varieties has to be replaced by a less satisfactory dust application because it is not possible to get into the orchard with a sprayer in late fall.

Depending on the number of sprays applied, the season, and the prevalence of canker infections in the orchard, control varies from 95 percent effective down to less than 50 percent.

The writers were interested in testing another material, maneb, for possible use where ziram had caused reported allergy conditions to an occasional worker or orchardist.

These tests were conducted in late September in two orchards. In orchard No. 1, near Peshastin, one plot was sprayed with 12 pounds per acre of ziram by hand, another was hand-sprayed with maneb (Dithane M-22). A third plot with ziram at 12 pounds per acre was applied 3X by "speed sprayer" and this plot was duplicated using maneb. In orchard No. 2, near Leavenworth, both the maneb and ziram plots were sprayed by hand gun from a stationary outfit, using 15 pounds per acre with Triton B 1956 wetter (2 ounces per 100 gallons).

The results in Table 1 are the averages of five replications. Each replicate consisted of one 40 pound lug of fruit per tree, stored at 34° to 38° F from picking time in October until January 20, 1958, and then stored at 60° until counting on February 4, 1958.

Table 1. Percent of Winesap apples showing bull's eye rot, February 4, 1958.

	Average percent bull's eye rot (5 replications)			
	Orchard No. 1		Orchard No. 2	
Treatment	Hand spray	Speed spray	Hand spray	Average
Ziram	5.3	2.6	20.2	9.4
Maneb	1.1	6.4	22.0	9.8
Unsprayed	10.4	--	36.3	23.4

The control from this one late spray following the heavy fall rains was not good in most cases. Maneb gave about 90 percent control in one hand-sprayed plot and about 60 percent control nearby with a speed sprayer. In the case of ziram the speed-sprayed plot was better. In all of our tests in earlier years we have found control with one late spray poor to good, with often no apparent reason for the difference. In general it is necessary to apply the earlier sprays also before the fall rains. In 1958 there was enough calyx end rot to indicate that an early summer spray would have paid well in these orchards.

The average indicates that maneb was about as effective as ziram and can be used in the same way as ziram.

WASHINGTON AGRICULTURAL EXPERIMENT STATIONS

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A DISORDER OF RANGPUR LIME ON SWEET ORANGE ROOTSTOCK

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Abstract

A disorder of a clone of Rangpur lime on sweet orange rootstock is described. Preliminary investigations suggest that the trouble is caused by a bud transmissible virus. This virus does not affect the growth of Rangpur lime rooted cuttings or sweet orange rooted cuttings. Failure occurs with this Rangpur lime as a scion on sweet orange, but not on sour orange rootstock.

In the course of growing trees for a collection of Rangpur limes at the University of California at Los Angeles, trouble was experienced with one clone budded on sweet orange seedlings and one budded on sour orange seedlings. The planting consisted of buds obtained from eight Rangpur lime varieties in the University orchard at Los Angeles. In the propagations made on sour orange rootstocks, trees budded from Red Ling Mung made very poor growth.

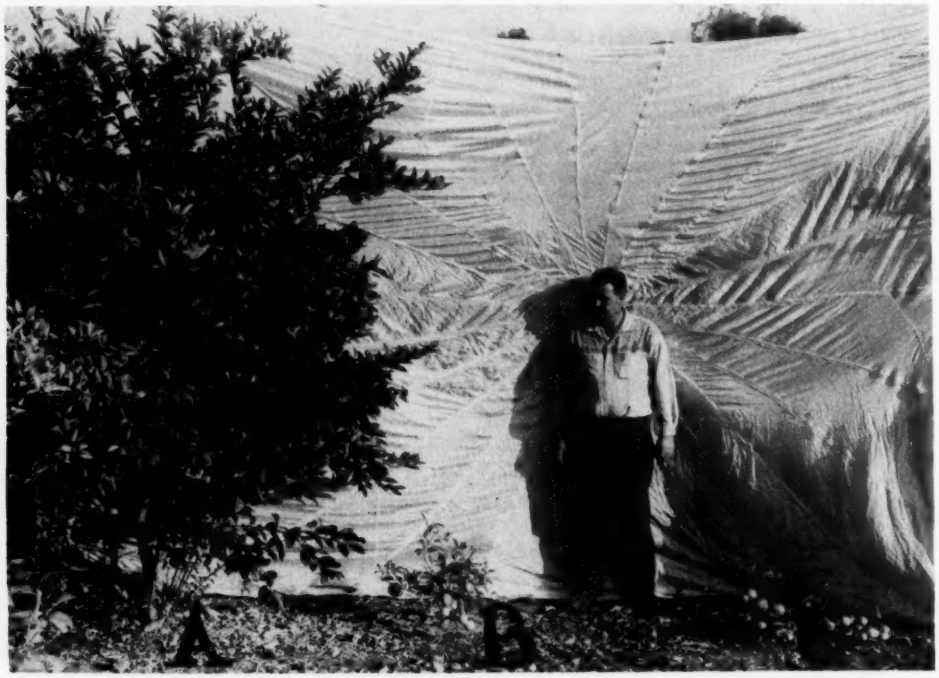

FIGURE 1. Growth of Rangpur lime selection C26-1 after 5 years on understocks of (A) sour orange, (B) sweet orange root with interstem of sour orange, (C) sweet orange.

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The Red Ling Mung was later shown by Wallace (2, 3) to be carrying tristeza and seedling yellows viruses. When the selections were propagated on sweet orange rootstocks, buds from a Rangpur lime tree of unknown origin (designated in this paper as C26-1) resulted in very weak trees. Tree C26-1 was infected with vein enation virus, but was free from tristeza and seedling yellows (2, 3). At Los Angeles the vein enation virus appears to be spreading, apparently by means of insect transmission, but the tristeza and seedling yellows in the same orchard have shown no evidence of natural spread over a 28-year period.

To determine if graft incompatibility was involved, scions from tree C26-1 were grafted on sweet orange, on sour orange, and on sweet orange with a sour orange interstem. These plants were grown by the cutting graft method described by Halma (1). The performance of the C26-1 selection on these different understocks is shown in Figure 1.

C26-1 on sweet orange root made normal growth for the first year, but in the second year chlorosis appeared accompanied by a heavy bloom and fruit set and little further growth. There was no beneficial effect from the sour orange interstem. C26-1 made satisfactory growth both on its own root and when budded on sour orange.

To determine whether a transmissible virus was involved, a plant of Red Ling Mung on sweet orange, which would normally grow well, was graft inoculated with a scion from tree C26-1. This resulting plant has developed chlorosis and is now making poor growth. A sweet orange rooted cutting inoculated with buds from tree C26-1 is making satisfactory growth.

The failure of Rangpur C26-1 on sweet orange is not due to vein enation virus because buds from other Rangpur lime trees carrying this virus have made satisfactory growth on sweet orange rootstocks. Thus it is concluded that this selection of Rangpur lime is infected with a virus which is distinct from any previously described from citrus.

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INCIDENCE OF SAVOY IN RELATION TO THE VARIETY OF SUGAR BEETS AND TO THE PROXIMITY OF WINTERING HABITAT OF THE VECTOR, PIESMA CINEREA

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Summary

Sugar beet savoy, a virus disease whose vector is the lacebug, Piesma cinerea Say, was found in far greater amount in an Ohio sugar beet field in 1952 and in a Minnesota field in 1955 than had previously been noted. The disease may be a more important factor in crop production than was formerly supposed. Those portions of the beet field that were nearest to unplowed areas, weedy lands, or wooded areas where the vector could find favorable winter quarters, showed the highest incidence of savoy. In the Ohio field, disease percentages starting at an average of 9.2 percent dropped at a rate of about 1 percent for each 100 feet of distance from the presumptive winter quarters of the vector; the drop in disease incidence in proceeding from presumptive winter quarters of the insect was at a lesser rate in the Minnesota field, dropping in 2900 feet from 20.0 percent to 1.4 percent.

Sugar beet varieties were found to react differentially to savoy. Resistance was shown by US 215 X 216, a leaf-spot-resistant variety, and by the hybrid between US 104 and US 400, a variety that combines resistance to leaf spot, curly top, and black root. If the disease were to become serious, possibility of effective control by breeding for savoy resistance is suggested.

INTRODUCTION

Sugar beet savoy, a virus disease transmitted by the lacebug, Piesma cinerea Say, has been known in the United States since 1890 when Arthur and Golden found affected plants in Indiana (1) and reported the disease as "not rare". They compared the appearance of leaves of diseased plants with that of savoy cabbage. The characteristic blackened rings of affected roots also were described. They assigned the disease to a bacterial organism but gave no exact evidence of pathogenicity. Out of 434 sugar beet roots sent to the Indiana Experiment Station for chemical analysis, 12 percent were found to be affected. Huston (6, 7, 8), in reports of production trials of sugar beets on Indiana farms, gave additional records on the occurrence of savoy, called by him "bacterial disease". The disease was widespread in Indiana in 1893, 1894, and 1895. It reduced root yields and seriously affected sucrose percentages. Cunningham (4) concurred in the earlier opinion that the disease was of bacterial nature. An organism was isolated from diseased root tissue but rigorous proof of pathogenicity was not given. The article has excellent photographs that identify the disease studied with what is today known as savoy. In 1902, Towar (9) reported the disease from Michigan under the name "leaf curl". His pictures and description leave no doubt that he was dealing with savoy. Incidence was given as from 1 to 3 percent, with late-planted beets showing only 0.5 percent. Three diseased sugar beet roots analyzed 6.9 percent sugar and 60.7 percent purity, whereas healthy plants taken from beside the diseased ones, and apparently of about the same size, showed 13.04 percent sugar and 76.3 percent purity.

Sugar beet plants showing symptoms typical of the disease as described by the earliest observers were collected in Michigan by Coons, Stewart, and Kotila, in 1923. From that time on, the disease has been found in various sugar beet growing areas. The virus nature of the disease and the fact that Piesma cinerea is vector of the virus were proved by tests started in 1934. These results were reported in 1937 (2) and the disease named savoy. A more complete account was given in 1950 (3).

The disease (Fig. 1) and its insect vector have been found in the United States in practi-

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FIGURE 1. Savoy of sugar beet. Early stage of infection with inner whorls of leaves showing symptoms. (Photo by J. E. Kotila, 1931.)

cally all sugar beet-growing States east of the continental divide; namely, Colorado, Wyoming, Montana, South Dakota, North Dakota, Texas, Nebraska, Kansas, Minnesota, Wisconsin, Indiana, Ohio, and Michigan. There is no record of the disease for Iowa or Illinois, but the insect occurs in these States and no doubt the disease also is present. Both the disease and its insect vector have been found in the experimental plantings of sugar beets at Arlington Farm, Virginia, and Beltsville, Maryland. Throughout the United States, Piesma cinerea is commonly found on Amaranthus retroflexus L., A. blitoides Wats., and on other species of Amaranthus. These are the favorite host plants. The insect will feed on sugar beets in the absence of its weed hosts, but it has only rarely been found on sugar beets in the field. Other plants, including grape, have been reported as hosts of P. cinerea.

In commercial sugar beet fields, plants affected by savoy have ranged from a trace to 1 or 2 percent, occasional fields showing in limited sections an incidence as high as 10 to 14 percent. Hildebrand and Koch (5) in 1942 reported the occurrence of savoy in many sugar beet fields in Ontario as a trace, but in several fields it occurred in higher percentages. One field was studied intensively. This showed an average incidence of 2.1 percent, but in portions of the field adjacent to weedy areas, the incidence of savoy was more than doubled. In one case, infection reached a maximum of 6.5 percent.

Observations made in the last decade have frequently shown an unevenness in the distribution of savoy in the field, with rather clear evidence of higher disease incidence in sugar beets growing near unplowed borders or in portions of the field near wooded areas or pasture land. Obviously, these areas would be favorable for hibernation of the insect vector.

In the earlier observations, it was not possible to relate disease incidence to sugar beet variety, since commonly the field was planted to a single variety. In a field planted with several varieties, as, for example, for a variety appraisal test, the situation very commonly was confused because the unplowed border or other overwintering habitat of Piesma cinerea was not so located with respect to the variety plots as to provide a dispersal front that would subject the varieties equally to the movement of the overwintering insects.

In 1952, and again in 1955, opportunities were afforded to make observations on two fields, each with a rather high incidence of savoy. In these fields, replicated plots of a number of sugar beet varieties had been planted. The plots were so located that apparently all varieties were equally exposed to the advance of Piesma cinerea from its overwintering quarters. In its habits, P. cinerea tends to be sedentary. It drops to the ground when disturbed, rather than fly away. In our experience, it was an extremely rare occurrence for any individual to take to its wings if disturbed. Any flight was very short. According to entomological records the insects have been known to move in great swarms. The special conditions or stimuli that bring about such movements are not known. The characteristic

sluggishness of the insect and its tendency to make few and short flights are believed to be factors in the localization of savoy as observed by us.

SAVOY AT PANDORA, OHIO

An agronomic evaluation test of leaf-spot-resistant and black-root-resistant varieties was conducted in 1952 on the Hiram Schutz farm near Pandora, Ohio, by J. G. Lill, in cooperation with the Buckeye Sugar Company and the Farmers and Manufacturers Beet Sugar Association. The experiment was a 9 X 9 Latin Square with plots four rows (28 inches) wide, 94 feet 9 inches long. The planting was made May 14 and the crop was harvested October 22. In August it was apparent that savoy was occurring in relatively high amounts throughout the field, 4.5 percent of the plants, on the average, being affected. In the period September 24 to October 18 a close check was made of all plants in the experiment and incidence of savoy noted for each plot. Sugar beet leaf spot (Cercospora beticola Sacc.) was absent, so the readings could be made very accurately. The field was slightly less than 4 acres in size and lay directly south of a paved State highway. At the east end, the entrance to the field was by a narrow grass-covered lane that separated the beet field from a small home orchard and garden plot. The layout of the experimental plot is shown in Figure 2.

It will be noted that the rows occupied by the different varieties were at right angles to the orchard and garden area -- the most likely overwintering habitat for the insects. The incidence of the disease in the experimental plot, as shown in Table 1, bears out this assumption. Replicate A had, as an average of all varieties, 9.2 percent savoy; Replicate B, 6.2 percent; and Replicate C, 8 percent. From Replicates D to I, the percentages of diseased plants fell off more or less regularly: 4.7, 3.8, 2.2, 1.6, 1.6, and 1.8 percent, respectively. The first 700 feet in the field showed a reduction in the incidence of savoy of about 1 percent for each 100 feet of distance from the likely source of insects.

The experiment, because of the arrangement of varieties and the apparent equality of exposures among varieties, also afforded an unusual opportunity to judge incidence of savoy in relation to sugar beet variety. The percentages of savoy among the nine varieties are shown in Table 2. From the data it appears that US 215 X 216 had significantly less savoy than any other variety. The other varieties in the test did not differ significantly from the general mean and probably not from each other. The experiment is of special interest because of the indication that factors for resistance to the savoy virus, or perhaps factors that condition nonattractiveness to the vector, may exist within the sugar beet complex.

SAVOY AT MANKATO, MINNESOTA

In 1955, a field of about 43 acres of American 3-S sugar beets was seen near Mankato, Minnesota, in which the incidence of savoy, although extremely high in the northern part, dropped to less than 1 percent at the south end of the field. The field was on the G. Arneman farm, and in it the American Crystal Sugar Company was conducting a small cooperative agronomic evaluation test of varieties resistant to both leaf spot and black root. The sugar beets in the field were attacked by Cercospora leaf spot, the injury being greatest in the nonresistant European Check variety. Incidence of savoy did not appear related to the leaf spot attack. If, however, the plants were severely injured by savoy, any leaves lost due to leaf spot were not effectively replaced, because of the dwarfing effects of the virus disease.

It was evident from inspection of the field that the heaviest incidence of savoy was in the northern portion where an unimproved road ran between the field and a high, wooded area -- a former river bank. Trees fringed the roadside opposite the field. On a part of the high bank there was a heavy growth of wild grape (Vitis vulpina L.) that extended nearly 150 feet along the north side of the field (Fig. 3).

Forty-eight sampling plots were set up in the sugar beet field, each plot being four rows (22 inches) wide by 50 feet long. The plots were 85 feet apart and were located on six bands that crossed the field at 50-, 450-, 750-, 1250-, 1950-, and 2900-foot intervals. The locations of the 48 sampling plots are shown in Figure 3. On September 17, 1955, the total number of beets in each plot and the number affected with savoy were determined. The savoy percentages found are shown on the field diagram. The average savoy percentage for the eight sampling areas of a given band is shown at the right margin. These averages reveal that in the band 50 feet from the uncultivated border area savoy affected approximately 20 percent of the plants, the average incidence dropping to 12.9 percent at the 450-foot band and to 10.8 and 9.2 percent, respectively, 750 and 1250 feet from the north edge of the field. At

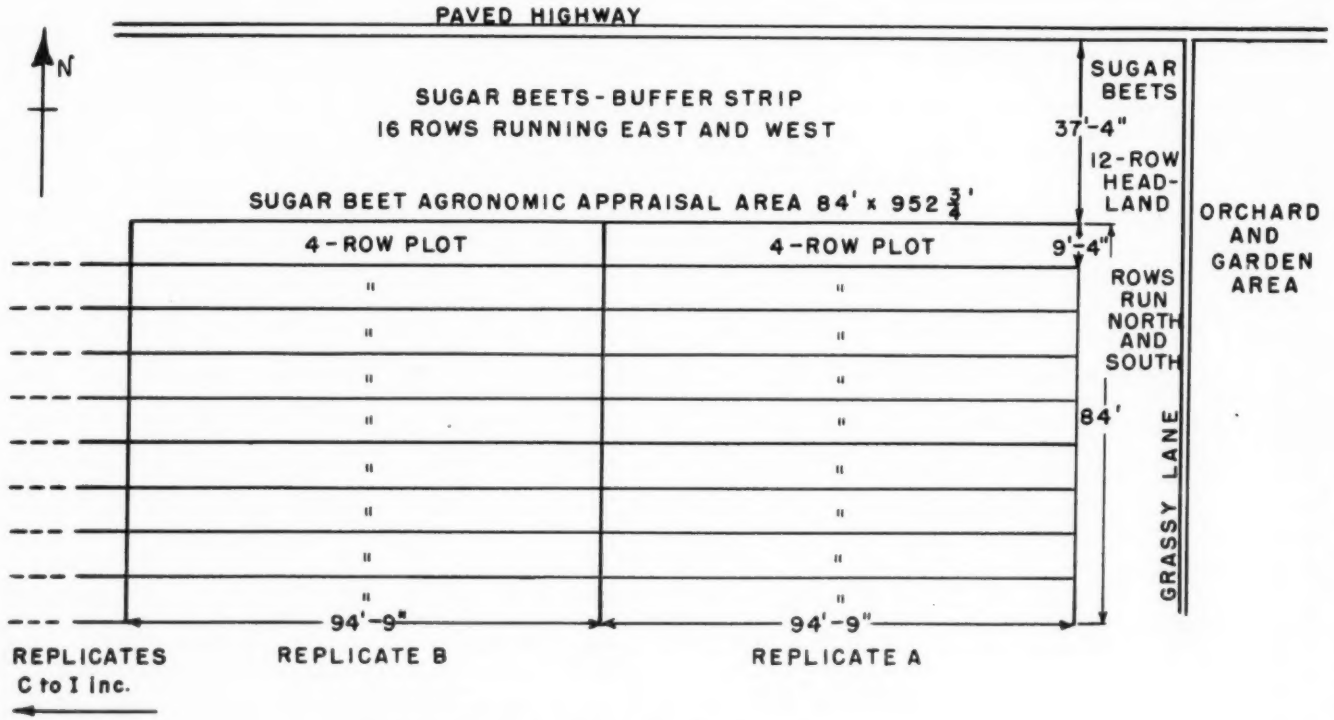

FIGURE 2. Diagram of sugar beet experimental field on the Hiram Schutz farm, Pandora, Ohio, in 1952. The probable source of overwintered adults of Piesma cinerea is the weedy orchard and garden plot at the eastern border of the experimental field.

Table 1. Agronomic appraisal test of sugar beet varieties on the Hiram Schutz farm, Pandora, Ohio, in 1952. Figures in parentheses are the savoy percentages as determined by counts in the period Sept. 28 to Oct. 18, 1952.

Replicates	Rows 1	2	3	4	5	6	7	8	9	Av.
A	SP 501712-02 (12.2)	Acc. 1192 (10.7)	SP 51107-01 (13.1)	SP 511701-00 (10.2)	US 216 X 226 (7.5)	Acc. 1191 (8.6)	US 215 X 216 (2.9)	SP 491704-02 (9.2)	Acc. 1205 (8.2)	(9.2)
B	511701-00 (7.4)	216 X 226 (6.1)	501712-02 (6.6)	51107-01 (8.4)	215 X 216 (0.9)	491704-02 (4.1)	1192 (3.9)	1205 (9.9)	1191 (8.6)	(6.2)
C	216 X 226 (6.3)	491704-02 (10.7)	1192 (11.5)	215 X 216 (5.8)	1205 (9.3)	511701-00 (7.2)	1191 (6.3)	51107-01 (6.0)	501712-02 (8.8)	(8.0)
D	1191 (2.2)	501712-02 (8.4)	1205 (2.4)	491704-02 (8.0)	511701-00 (5.4)	1192 (4.4)	51107-01 (3.7)	216 X 226 (5.1)	215 X 216 (2.8)	(4.7)
E	1205 (3.8)	51107-01 (3.8)	491704-02 (6.3)	1191 (3.1)	501712-02 (4.1)	215 X 216 (0.3)	511701-00 (4.3)	1192 (4.4)	216 X 226 (4.4)	(3.8)
F	215 X 216 (1.5)	1205 (3.4)	216 X 226 (0.6)	1192 (1.5)	1191 (1.2)	51107-01 (0.7)	491704-02 (3.5)	501712-02 (3.9)	511701-00 (3.9)	(2.2)
G	491704-02 (1.3)	215 X 216 (0.5)	1191 (0.9)	1205 (2.6)	1192 (1.4)	501712-02 (0.9)	216 X 226 (2.1)	511701-00 (3.3)	51107-01 (0.9)	(1.6)
H	1192 (0.9)	511701-00 (3.9)	215 X 216 (0.0)	216 X 226 (1.6)	51107-01 (0.9)	1205 (1.8)	501712-02 (1.4)	1191 (0.8)	491704-02 (2.8)	(1.6)
I	51107-01 (3.4)	1191 (1.5)	511701-00 (3.8)	501712-02 (0.8)	491704-02 (1.2)	216 X 226 (0.4)	1205 (1.5)	215 X 216 (0.6)	1192 (3.2)	(1.8)

Table 2. Sugar beet savoy percentages in variety test at Pandora, Ohio, in 1952; data of the 9 X 9 Latin Square as given in Table 1 are distributed by variety and replication.

Variety	Description	REPLICATION A	B	C	D	E	F	G	H	I	Av.
US 215 X 216	**Leaf Spot Res.**	2.9	0.9	5.8	2.8	0.3	1.5	0.5	0.0	0.6	1.7
Acc. 1191	LS & BR Res.	8.6	8.6	6.3	2.2	3.1	1.2	0.9	0.8	1.5	3.7
US 216 X 226	**Leaf Spot Res.**	7.5	6.1	6.3	5.1	4.4	0.6	2.1	1.6	0.4	3.8
SP 51107-01	US225MS X 201	13.1	8.4	6.0	3.7	3.8	0.7	0.9	0.9	3.4	4.6
Acc. 1192	LS & BR Res.	10.7	3.9	11.5	4.4	4.4	1.5	1.4	0.9	3.2	4.7
Acc. 1205	European Ck.	8.2	9.9	9.3	2.4	3.8	3.4	2.6	1.8	1.5	4.8
SP 491704-02	US225MS X 226	9.2	4.1	10.7	8.0	6.3	3.5	1.3	2.8	1.2	5.2
SP 501712-02	US225 X Syn.	12.2	6.6	8.8	8.4	4.1	3.9	0.9	1.4	0.8	5.2
SP 511701-00	Syn. var.	10.2	7.4	7.2	5.4	4.3	3.9	3.3	3.9	3.8	5.5

General Mean 4.3
L.S.D.(5% point) 1.5

1950 feet from the presumptive source of Piesma cinerea, the incidence of savoy remained fairly high (7.3 percent), but at the extreme south of the field, a distance of 2900 feet, the disease occurred in a low percentage (1.4 percent).

Attention is called to the very high incidence of disease that is shown in sampling areas 4, 5, and 6 (as numbered from the east) of the band 50 feet from the north border. Stewart and Schneider, as a result of their observations on the field, felt that there was a strong possibility that the wild grape (Vitis vulpina) was in some way, possibly as a reservoir of the virus, concerned with the high incidence of savoy. Clones of these wild grapes have been established, and tests are under way to determine whether the virus occurred in this host of Piesma cinerea. It will be noted that the high incidence of savoy carried nearly halfway into the field along a swath of about 138 rows (Fig. 3).

An agronomic evaluation test of eight sugar beet varieties, conducted in the field as an 8 X 8 Latin Square, was located at the north edge of the field at about the point of greatest savoy incidence. The plots were four rows wide (22 inch-rows) and 35 feet long. All plants in the plots were counted on September 17, 1955, and the number of plants with savoy determined. The arrangement of varieties in the experimental area and the percentages of savoy are shown in Table 3. The data of this Table are collected in Table 4 by variety and by replication. Among the varieties, the average percentage of plants affected by savoy ranged from 14.3 percent for Accession 1360 (the hybrid of US 104 and US 400), which combines resistance to curly top, leaf spot, and black root, to 28.5 percent for European Check which is highly susceptible to all these diseases. Both values differ significantly from the general mean of the experiment. These savoy readings are the extremes of the test and were consistent throughout the eight replications. The other varieties constitute a group falling between these extremes and do not differ significantly from the general mean. With the exception of Accession 1320-A, these varieties are rather closely related, so that concordance in their behavior with respect to disease reaction could be expected.

This experiment, like the one at Pandora, Ohio, is noteworthy in showing that a relatively heavy incidence of savoy can occur in sugar beet fields. Yield and sucrose data were taken by the American Crystal Sugar Company to evaluate the varieties. The attack by leaf spot was very severe and it is not possible to separate out the effects attributable to savoy alone. For this reason, data on yield and sucrose percentages are not included in this report. It was very clear that root yields and sugar production from the field were greatly below the field potential. The sucrose percentage of the European variety used as check was lower by about

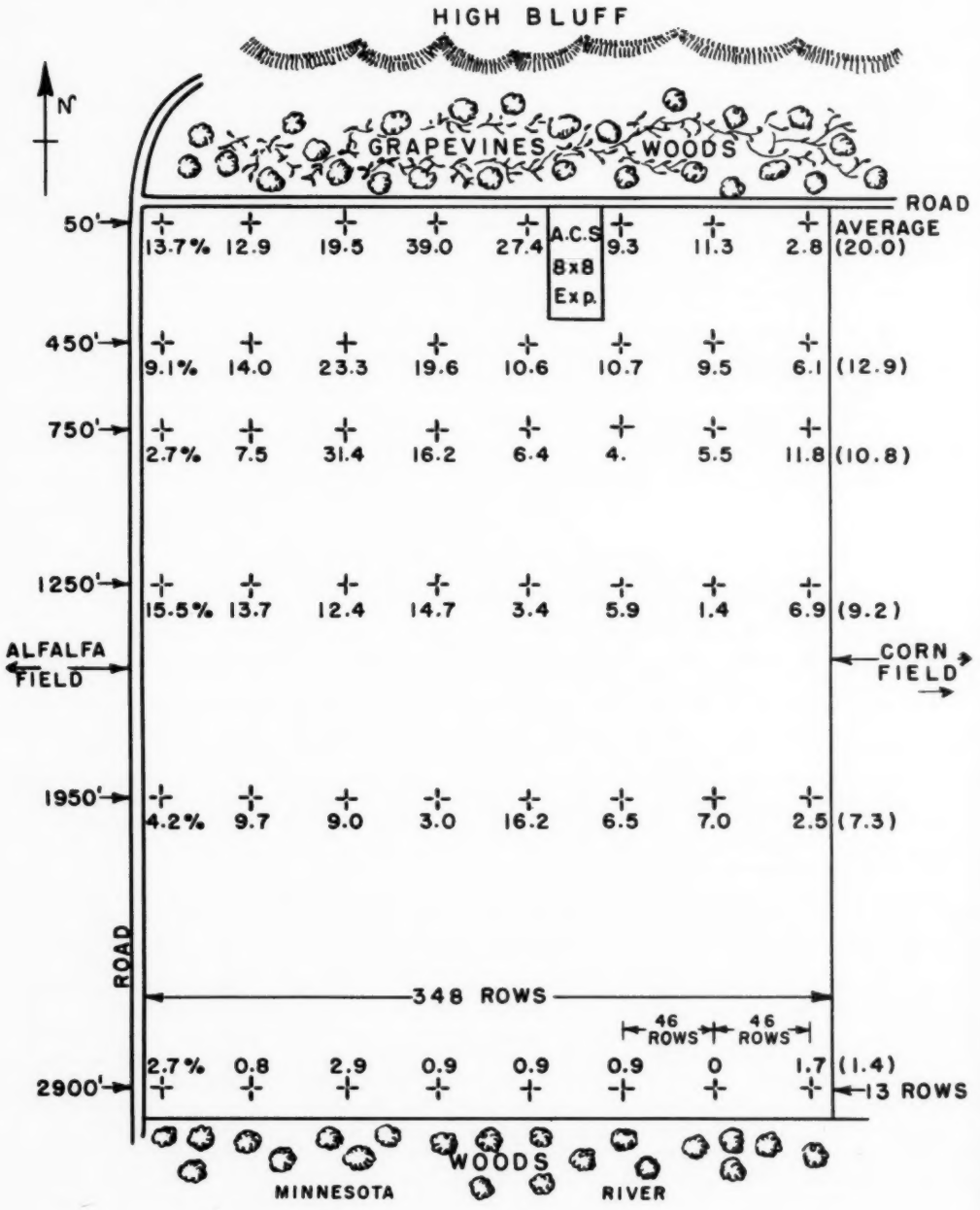

FIGURE 3. Sugar beet field on G. Arneman farm near Mankato, Minnesota, that showed high percentages of savoy in north portion; highest disease incidence occurred in a band of rows near the center of the north end and extended south almost halfway in the field.

Table 3. Agronomic appraisal test of sugar beet varieties on the B. Arneman farm, Mankato, Minnesota. Figures in parentheses are savoy percentages as determined by counts made on September 17, 1955.

Replicates	Rows 1	2	3	4	5	6	7	8	Av.
A	Acc.1320-A (27.8)	Acc.1352 (31.3)	Acc.1327 (30.6)	Acc.1353 (20.7)	Acc.1350 (26.2)	Acc.1351 (32.0)	Amer. 3-S (29.9)	Acc.1361 (19.8)	(27.3)
B	Acc.1351 (25.5)	Acc.1327 (31.8)	Acc.1320-A (20.8)	Acc.1350 (27.5)	Acc.1361 (17.4)	Acc.1352 (22.4)	Acc.1353 (34.3)	Amer. 3-S (46.5)	(28.3)
C	Acc.1361 (14.5)	Acc.1353 (20.0)	Acc.1350 (27.2)	Acc.1351 (24.4)	Acc.1352 (28.7)	Amer. 3-S (24.1)	Acc.1320-A (37.4)	Acc.1327 (48.2)	(28.1)
D	Acc.1352 (10.1)	Acc.1320-A (11.8)	Acc.1351 (11.8)	Acc.1361 (13.8)	Amer. 3-S (21.6)	Acc.1327 (39.0)	Acc.1350 (25.7)	Acc.1353 (19.5)	(19.2)
E	Acc.1350 (20.8)	Amer. 3-S (10.9)	Acc.1353 (11.8)	Acc.1320-A (13.3)	Acc.1351 (33.9)	Acc.1361 (16.5)	Acc.1327 (20.8)	Acc.1352 (18.7)	(18.3)
F	Amer. 3-S (10.2)	Acc.1350 (8.3)	Acc.1361 (10.4)	Acc.1352 (11.0)	Acc.1327 (15.3)	Acc.1352 (9.4)	Acc.1351 (7.5)	Acc.1320-A (17.7)	(11.2)
G	Acc.1353 (12.7)	Acc.1361 (10.1)	Amer.3-S (22.2)	Acc.1327 (20.3)	Acc.1320-A (11.1)	Acc.1350 (12.5)	Acc.1352 (13.4)	Acc.1351 (17.1)	(14.9)
H	Acc.1327 (21.8)	Acc.1351 (16.7)	Acc.1352 (6.9)	Amer.3-S (13.4)	Acc.1353 (8.0)	Acc.1320-A (10.9)	Acc.1361 (11.9)	Acc.1350 (12.0)	(12.7)

Table 4. Sugar beet savoy percentages in variety test at Mankato, Minnesota, in 1955; data of the 8 X 8 Latin Square as given in Table 3 are distributed by variety and replication.

Code	Variety	Description	Replication A	B	C	D	E	F	G	H	Av.
8	Acc. 1361	LSR-CTR X US400	19.8	17.4	14.5	13.8	16.5	10.4	10.1	11.9	14.3
4	Acc. 1353	Inc.52108-0 BRR	20.7	34.3	20.0	19.5	11.8	9.4	12.7	8.0	17.0
2	Acc. 1352	Inc.EL 1012 BRR	31.3	22.4	28.7	10.1	18.7	11.0	13.4	6.9	17.8
1	Acc. 1320-A	US225MS X 201B	27.8	20.8	37.4	11.8	13.3	17.7	11.1	10.9	18.8
5	Acc. 1350	Inc.53AB1-0 BRR	26.2	27.5	27.2	25.7	20.8	8.3	12.5	12.0	20.0
6	Acc. 1351	US 401	32.0	25.5	24.4	11.8	33.9	7.5	17.1	16.7	21.1
7	Amer. 3-S	Local	29.9	46.5	24.1	21.6	10.9	10.2	22.2	13.4	22.4
3	Acc. 1327	European Check	30.6	31.8	48.2	39.0	20.8	15.3	20.3	21.8	28.5
									General Mean		20.0
									LSD (5% Point)		6.0

3 percentage points than the general run of the other varieties. It is known that severe leaf spot attack can reduce sucrose percentages significantly, even as much as 2 percentage points. In this case, it would appear that the savoy augmented the loss from leaf spot and the combination of the two diseases produced heavy damage to the crop.

DISCUSSION

So far as is known, savoy is contracted by sugar beets only as a result of feeding by viruliferous adults of Piesma cinerea. The assumption that the insect winters in grassy, wooded, or shrubby areas seems well substantiated by the higher incidence of savoy in those portions of sugar beet fields that have close proximity to what, presumptively, are suitable winter quarters for the vector of the savoy virus. In Europe, P. quadrata Fieb., the beet leaf bug, is known to be the vector of a related disease, sugar beet Kräuselkrankheit. Studies by Wille (10) have shown that P. quadrata overwinters in unplowed ground, in stubble, in crevices in the bark of trees, or wherever it can get some protection. The viruliferous adults of P. quadrata move to the sugar beet fields in the spring, particularly in May, initiating Kräuselkrankheit. The observations on P. cinerea indicate a close similarity in the habits of these related species.

It is becoming increasingly evident that many sugar beet fields in the United States, east of the Rocky Mountains, are experiencing severe losses from savoy. It was previously believed that the disease occurred only in minor amounts and that increased growth of the adjacent nonaffected plants compensated for the scattered savoy plants. With disease incidence of the type described here, and with the known depressing effects of savoy on both growth and sucrose percentage, the crop losses from savoy may be serious. Both the insect vector and virus have extremely wide distribution. All Piesma cinerea collections tested so far have been found to have some viruliferous individuals, no collection having failed to give one or more savoy-affected plants if the adults were caged in groups of five or more on healthy plants. Collections made a mile or more from the nearest sugar beet or mangel-wurzel field, and equally far from a home garden that might be growing red garden beets or chard, also were found to contain viruliferous individuals. In the humid area, the percentages of savoy in sugar beets seem closely related to the proximity of the field to suitable quarters in which the insect may hibernate.

Control of the disease in the United States probably can be accomplished very simply by sanitation measures directed against the insect or by locating the sugar beet field at a distance of a few hundred feet from any woodlot or uncultivated land that could serve as an in-

sect source. Some crop other than the sugar beet may be grown next to the fence row, woodlot, or other potential source of the insect vector of savoy. It does not seem that the intensive measures used in Europe to control Kräuselkrankheit of sugar beets through the destruction of the overwintered vector of the virus are called for at present to control savoy in sugar beet fields. In Europe, a trap crop consisting of a few rows of sugar beets is planted as early in the spring as possible around a prospective beet field and before the main part of the field is sown. In early May, the trap crop of sugar beet seedlings, with the overwintered Piesma quadrata adults that it attracts, is plowed under; then the regular beet crop is planted. Apparently the plowing under of the trap crop so reduces the population of the vector as to make the field safe for sugar beets. Some loss accrues in this control measure because of the delay in planting the regular crop of sugar beets, but this is slight compared to the almost complete destruction that, in the Kräuselkrankheit zone of Europe, can be suffered by sugar beet fields to heavily invaded by viruliferous P. quadrata. More recently, systemic insecticides have been used on the trap crop to kill the insects and thereby permit more prompt planting of the main crop of sugar beets.

The finding reported here, that certain varieties of sugar beet consistently show less savoy than others, indicates that if savoy were to become a serious factor in crop production, control probably could be effected by breeding resistant varieties. The high incidence that may occur shows that savoy of sugar beets should be recognized as a potentially dangerous sugar beet disease and observations on it should be continued.

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TOBACCO AND SUGAR CROPS BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

OCCURRENCE OF CORN STUNT VIRUS IN CENTRAL AMERICA AND COLOMBIA

Donald L. Smith[1] and John S. Niederhauser[2]

During 1956 and 1957 diseased corn plants with symptoms characteristic of the corn stunt virus strains Rio Grande and Mesa Central were observed in the following locations:

Country	Location
Guatemala	Guatemala City
	Barcena
	Cuyuta
El Salvador	San Andres
Honduras	Comayagua
Nicaragua	Managua
	Rivas
Costa Rica	Las Delicias
	Turrialba
Panama	Divisa
Colombia	Palmira

Identification was based on symptomatology at the time of flowering. Of the two strains, Mesa Central predominated in both years, although in 1957 the Rio Grande strain tended to increase slightly at locations in Guatemala and Nicaragua. The prevalence of infection varied considerably with the location and with the type of corn at each location.

At all locations where breeding programs were conducted the percentage of infected plants was higher in the inbred lines than in single crosses, hybrids, or varieties. Both the prevalence and the severity of infection differed considerably in the different lines. Many lines were apparently free of the virus, while in others up to 75 percent of the plants were infected. In a double cross production field (Rocamex H-501) at Managua, 100 percent of the male plants and approximately 80 percent of the female plants were infected. The male plants were so seriously affected that pollen production was impaired and the field was lost for seed production. At the same location, however, less than 5 percent of the plants were infected in a field of the same double cross.

It is clear that the corn stunt virus can be very destructive in some kinds of corn presently being grown in these Central American countries and in Colombia. However, as there appear to be a number of sources of resistance, it seems probable that the disease can be kept in check by breeding for resistance.

MEXICAN AGRICULTURAL PROGRAM OF THE ROCKEFELLER FOUNDATION

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THE 1957 SOIL-BORNE WHEAT MOSAIC EPIPHYTOTIC IN KANSAS[1]

Webster H. Sill, Jr., and Claude L. King[2]

Summary

An epiphytotic in winter wheat in Kansas incited by soil-borne wheat mosaic virus is reported. Approximately 347,000 acres of wheat were diseased. Estimated losses were 2,082,000 bushels, worth approximately $3,950,000. Estimates of loss in diseased fields varied from 5 to 12 bushels per acre. In all tests in infested soil the highly resistant variety Concho was first in yield even though badly lodged. The resistant variety Comanche was second in most cases.

A severe epiphytotic of soil-borne wheat mosaic occurred in eastern Kansas winter wheat during the 1956-57 season. This virus disease was observed by the junior author and Dr. Hurley Fellows[3] in Kansas about 1949 and was first demonstrated to be caused by a soil-borne virus by McKinney (5). The first known major epiphytotic in Kansas occurred in 1952 (1). A second more severe outbreak developed in 1954 when losses reached 3 million dollars (2). The 1957 epiphytotic has convinced agricultural workers in the area that this will continue to be an important wheat disease in Kansas until resistant varieties are grown more widely than at present. To meet this threat the Kansas Agricultural Experiment Station in 1957 recommended the soil-borne mosaic-resistant varieties, Comanche and Concho, for production in virus-infested soils. These varieties were reported earlier as highly resistant in Kansas (6).

The seasonal development of disease and the symptomatology followed the pattern of other years (1), which is quite similar to that described by Koehler et al. (3) in Illinois except that no wheat rosetting has been observed in Kansas in any variety.

Webb (7), McKinney (4), and Koehler et al. (3) have reported a mild winter and a long cool, moist growing season in fall and spring as favorable for disease development. The 1956-57 winter wheat growing season fulfilled these requirements. The long cool growing periods were present and moisture was adequate in eastern Kansas in the fall, with good rains in October and early November, and abundant to excessive rainfall during the spring. The winter was dry and mild, with only two brief periods of subzero weather and no winter-killing. Soil-borne wheat mosaic symptoms began to appear in late February. Heavy cool spring rains began about the middle of March. By March 20 symptoms were very severe throughout eastern Kansas and it was obvious to the authors and others that the largest acreage of infected wheat on record was involved. The cool, cloudy, moist weather persisted until about April 15. At that time the infected fields of wheat were deteriorating each day and the symptoms were at their worst.

About April 15 a warm period of several weeks' duration commenced. By May 1 all plants in infected fields had recovered from their bronze to yellow-green chlorosis and were nearly a normal green although mosaic stunting and mottling persisted. This elimination of chlorosis at temperatures above approximately 60° F has been reported by many workers as a characteristic of this disease. During May and June the wet conditions returned and persisted with temperatures well below normal in most parts of the State. These cool wet conditions continued until harvest and caused the development of extremely tall, rank wheat which lodged very badly during frequent rains and high winds. The mosaic and mottling symptoms persisted, but the yellowing of early spring did not return. So much lodging

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[1]Contribution No. 515, serial No. 666, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan.

The authors acknowledge with appreciation the help of many Kansas county agents, particularly the following men who compiled yield data: Edwin Hedstrom, Walter Campbell and Jerry Hope.

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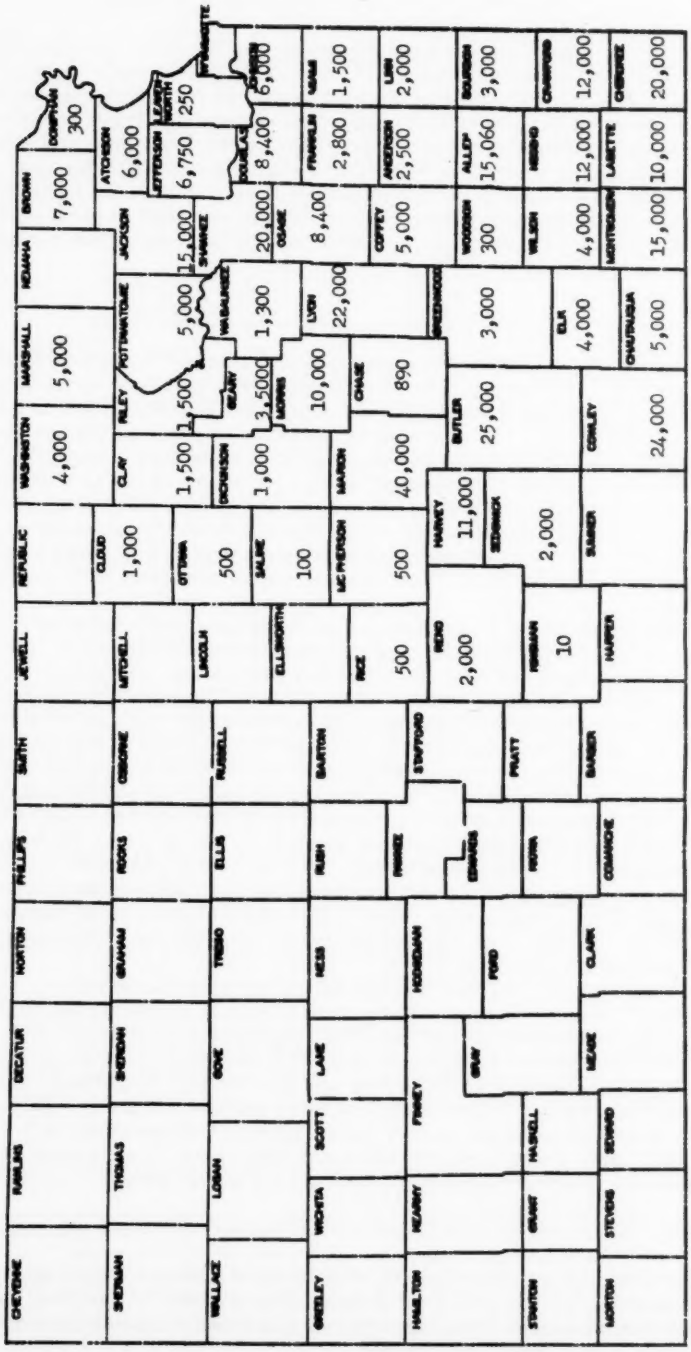

FIGURE 1. Location of the estimated 347,000 acres of Kansas wheat having soil-borne mosaic in 1957.

occurred that it was difficult to separate mosaic losses from those caused by lodging and other conditions.

A survey made with the help of Kansas county agents showed that soil-borne wheat mosaic occurred in 50 eastern Kansas counties (Fig. 1) in 1957. Approximately 347,000 acres of wheat were diseased, representing about 25 percent of the acreage in these counties. The average yield reduction in diseased fields, as estimated by county agents, was 16.5 percent or approximately 6 bushels per acre. This was an estimated loss of 2,082,000 bushels, valued, at current prices, at approximately $3,950,000. The estimate is probably low because of the extreme difficulty in separating the various loss factors present. Farmers' loss estimates were uniformly higher, averaging about 10 bushels per acre. Four county agents who actually took comparative yield data in diseased and non-diseased areas reported loss estimates which varied from 5 to 12 bushels per acre.

In Osage County the county agent, Walter Campbell, inadvertently planted his wheat variety demonstration plots in soil uniformly infested with soil-borne wheat mosaic virus. Even though the resistant Concho and Comanche varieties were badly damaged by lodging, they still outyielded the other varieties, all of which are susceptible in varying degrees. The yields are presented in Table 1. Lodging data were not recorded.

Table 1. Wheat yields in bushels per acre in 1957, taken from County variety demonstration plots planted on soil uniformly infested with soil-borne wheat mosaic virus.

	Marshall County			Osage County	
Variety	Rank	Yield	Percent lodged[a]	Rank	Yield
Concho	1	44	87	1	35
Ponca	2	43	50	5	22
Comanche	3	38	75	2	34
Triumph	4	37	0	6	21
Wichita	6	33	75	7	21
Pawnee	7	31	25	4	25
Tenmarq	8	30	75	8	19
Bison	9	29	25	3	27
Kiowa	5	36	50	9	18
Iowin	10	27	50	11	16
Cheyenne	11	23	75	10	18

[a]The harvest and preharvest seasons were unusually wet and windy in 1957 which accounts for the large amount of lodging. Lodging was present in the Osage County plots but the data were not recorded.

Edwin Hedstrom, Marshall County Agent, purposely planted one of his 1957 wheat variety demonstration plots in soil uniformly infested with soil-borne wheat mosaic virus. The yield and lodging data are presented in Table 1. Concho was higher in yield than any other variety although badly lodged. The susceptible variety Ponca was second and the resistant variety Comanche third. Triumph, an early susceptible variety which did not lodge, was fourth. A severe epiphytotic of leaf rust (Puccinia recondita) occurred in these plots. The variety Ponca probably was near the top in yield because it was the only variety highly resistant to leaf rust. Three varieties had been grown in these same plots during the three preceding years, 1954-1956. Soil-borne mosaic was severe in only one of these years. The 3-year average yields were: Concho 42, Ponca 38, and Pawnee 36, bushels per acre. Mr. Hedstrom had plots in three other infested fields in 1955. This was not a severe mosaic year and symptoms were very mild or absent. The average yield differences in favor of Concho over the next highest varieties were 5.5 bushels per acre. However, Concho did not outyield several of the test varieties when planted in noninfested areas. These data would indicate that soil-borne mosaic may cause considerable damage even when symptoms are mild or absent.

In 1957 the Dickinson County Agent reported that conditions were extremely favorable for the early variety, Triumph. In this county Triumph outyielded Concho by 3 bushels per acre on infested land. Even so, when all of eastern Kansas was considered, Concho and Comanche were the highest yielding varieties in 1957 on soil-borne-mosaic virus infested soil.

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OAT CHLOROSIS IN SOUTH CAROLINA IN 1957[1]

W. P. Byrd, R. W. Earhart, and E. B. Eskew[2]

Abstract

A chlorosis of oats appeared in the Piedmont of South Carolina during March and April of 1957. Differences in varietal reactions to this chlorosis were evident in the oat agronomic nurseries at Clemson and yields of affected varieties were reduced. Varieties least affected were: Arlington, Southland, LeConte, Lemont, and C. I. No's. 6904, 6576, 6980, 7129, and 7226. Most severely affected varieties were: Alamo, Victorgrain 48-93, Midsouth, and C. I. No's. 7253, 7231, and 7219. Production losses were estimated by comparing the yields of Victorgrain 48-93 and Arlington for 3 years (1954-56), when chlorosis was not present, with the results from three tests in 1957 where chlorosis was present.

During late March and early April of 1957 a chlorosis of oats appeared widespread in the Piedmont area of South Carolina. The presence of this disease had not previously been determined as important, and initial investigations proved it to be distinct from a chlorosis of oats and other small grains in the Coastal Plains which has been associated with mineral element nutritional unbalance on soils of a high pH. The symptoms of this present chlorosis are a yellow or golden color, either inter-veinal or solid on the newly formed leaves. Seriously affected plants are greatly stunted and in some cases are killed or fail to head. These symptoms generally continue to develop throughout the remainder of the life of the affected plant and in many cases affect the glumes.

In many of the fields observed there was a general distribution of the chlorosis over the entire field. Often, however, normal plants were growing adjacent to chlorotic plants even in the most severely affected areas.

A recent report from Georgia[4] designated a similar chlorosis in oats as "yellow leaf" and attributed it to an aphid-transmissible virus. Unpublished data from South Carolina[3] show that under greenhouse conditions the chlorotic symptoms could be duplicated by growing susceptible oat plants in soil infested with isolates of Helminthosporium spp. obtained from plants manifesting this chlorotic situation.

This chlorosis was severe in the breeding nursery at Clemson, which afforded an opportunity to study oat varietal reaction. The reactions, expressed as the percentage of foliage showing chlorosis, are reported in Tables 1 and 2 which present data for the oat varieties included in the Uniform Central Area Winter Oat Nursery and the Uniform Southern Winter Oat Nursery grown at Clemson in cooperation with the United States Department of Agriculture. Arlington, Southland, and several selections expressed little chlorosis, whereas Alamo, Victorgrain 48-93, Midsouth, Floriland, Appler, Seminole, and several selections were highly susceptible. With the exception of the variety Southland, the more "upright" or "spring-type" varieties had the most pronounced chlorosis. It was also apparent that more of the varieties included in the Uniform Central Area Nursery possessed higher degrees of resistance than did the varieties from the Southern Nursery. The average percentage of chlorosis was higher and the average yield lower in the Southern Nursery as compared with the Central Nursery. It is interesting to note that Midsouth, a "Victoria" blight resistant selection from Victorgrain 48-93, had about the same reaction as its parent variety.

Although differences in yield cannot be wholly attributed to the effect of this chlorosis, it is apparent that the varieties with the highest amounts of yellowing were consistently among

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[1]Technical contribution No. 281, South Carolina Agricultural Experiment Station.

[2]Associate Agronomist, Plant Pathologist, and Associate Agronomist, South Carolina Agricultural Experiment Station, respectively.

[3]Earhart, R. W., and M. H. Thomas, Jr. 1958. A cause of oat yellowing. Presented to Southern Section of American Phytopathological Society, Little Rock, Arkansas, Feb. 3, 1958.

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Table 1. The reaction to oat chlorosis of certain varieties and selections of oats in the Central Area Winter Oat Test and the subsequent test weights and yields at Clemson, South Carolina in 1956-57.

C. I.	Variety or Selection	Percent Chlorosis (4-12-57)	Wt./Bu. (Pounds)	Yield (Bu./A.)
7225	C.I. 4383 x Bonda: Md. '56-54	4	29.0	88.2
7227	C.I. 3717 x Sib. 4316 x ($C1^2$ - SF): Md. 56-98	4	34.5	75.1
4657	Arlington	tr	31.0	70.6
6904	L-V x Forkedeer: Purd. 392A-28-5	tr	29.5	70.3
7226	C.I. 3717 x Sib. 4316 x ($C1^2$ - SF): Md. 56-97	tr	29.5	69.1
7228	C.I. 3717 x Sib. 4316 x ($C1^2$ - SF): Md. 56-94	3	31.0	67.6
6576	Atlantic x ($C1^2$ - SF): Md. Sel.	tr	32.5	66.4
7230	Wintok x S.F.: Ga. H 1617-x-2	1	29.5	59.4
6994	Tennex x Victoria/Haj-Ban: Tex. 119-50-8	4	31.5	55.6
7229	Arl.-Del./Trisp. x Bond - Fulg./Vg.: Coker 56-38	5	29.5	46.0
7150	Forkedeer x Fla. 19-11: Fla. Ab. 209	4	29.5	42.7
6993	Fulwin Comp: Tex. 152-50-17	5	27.0	41.7
7218	(Vg - Fulwood) x (Coker 53-29): Coker 56: 18	10	29.0	35.7
7224	C.I. 4383 x Bonda x Landhafer: Ark. C4-5-1	2	26.5	32.7
7231	Carolina Red x ($C1^2$ - SF): Delta Sel. 5216-6	27	27.5	27.3
7125	Victorgrain 48-93	23	27.7	26.5
1815	Appler	15	23.0	22.9
1815	Appler	20	20.5	16.6

the lowest in yield.

LeConte, Lemont, C. I. 6980 (Ballard Selection), and C. I. 7129 (Wintok x Atlantic), although not included in the oat nurseries summarized in Tables 1 and 2, showed no chlorosis and produced normal growth in the Clemson Oat Nursery.

Table 3 presents a comparison of several oat varieties which were planted at two different dates: on October 15, 1956, which is considered the optimum planting date for oats in the Clemson area, and on November 1, which is later than the optimum planting time. Chlorosis was more severe on early planted oats; however, the detrimental effect of late planting appeared to offset any gain from the reduction in the amount of chlorosis.

Table 2. The reaction to oat chlorosis of certain varieties and selections of oats in the Uniform Southern Winter Oat Experiment and the subsequent test weights and yields at Clemson, South Carolina in 1956-57.

C. I.	Variety or Selection	Percent Chlorosis (4-12-57)	Wt./Bu. (Pounds)	Yield (Bu./A.)
4657	Arlington	tr	30.0	85.1
7259	(Let./Cl^2 - SF) x Arlington	2	29.5	72.1
7261	Atlantic x Cl^2/SF	4	30.0	69.1
7134	Trisp./Cl^2 - SF x Atl./Cl^2 - SF	3	27.5	61.1
7258	" " " "	5	28.0	58.8
7255	Fla. 167/SF-C1 x C.I. 5208/C.I. 5919-Canuck	6	25.5	50.5
	Fulwood (Local Check)	4	25.0	50.3
5207	Southland	tr	26.0	49.8
7260	Atlantic x Cl^2/SF	3	27.5	49.5
7229	Arl. - Del./Trisp. x B-F/Vict.: Coker 56-33	9	30.0	49.4
7218	Victorgrain/Ful. x (53-29): Coker 56-18	14	30.0	39.1
5924	Seminole	10	24.0	36.9
1815	Appler	12	21.0	34.4
1815	Appler	10	21.5	30.9
4653	Delair	6	26.0	30.8
7219	Victorgrain x Bonda-HJ/SF	21	27.0	26.3
7125	Victorgrain 48-93	19	27.0	25.1
6977	Midsouth	19	27.0	24.8
5371	Alamo	37	27.0	23.2
7236	Victorgrain 48-93 x BR-HJ/Landhafer	9	25.5	22.8
7148	O-200-10 x Southland	12	27.0	22.7
6588	Floriland	15	23.0	22.5
7149	Southland/Landhafer x Fla. 19-11	4	24.0	19.7
6600	Sunland	6	22.5	10.9
7253	Delair x Bonda-HJ/SF	45	-	9.4

Table 3. Comparison of the yields and percent chlorosis of certain varieties planted at different dates in 1956-57.

C. I. Variety	Planted Oct. 15		Planted Nov. 1	
	Percent Chlorosis	Yield (Bu./A.)	Percent Chlorosis	Yield (Bu./A.)
4657 Arlington	trace	76.4	trace	77.8
7229 Coker 56-38	14	44.9	7	47.7
7218 Coker 56-18	34	49.2	12	37.4
7125 Victorgrain 48-93	36	37.0	21	25.8
6977 Midsouth	43	32.9	19	23.2

Table 4. The relative yields of Arlington and Victorgrain 48-93 in the presence of chlorosis in 1956-57 compared with the previous 3-year period when chlorosis was not observed.

Variety	Yield (Bu./A.) (Ave. 3 yrs.) 1954, 55, 56	Yield (Bu./A.) (Ave. 3 tests) 1957
Arlington	82.9	77.7
Victorgrain 48-93	89.1	29.5
Victorgrain as percent of Arlington	107%	38%

The variety Victorgrain 48-93, which manifested severe chlorotic symptoms, is planted on the greater part of the oat acreage in South Carolina. Therefore, it is interesting to compare the relative yields of this variety with Arlington, which had only trace amounts of chlorosis. In the 3 years prior to 1957, the average yield of Victorgrain 48-93 was 7 percent higher than that of Arlington in the Clemson tests (Table 4). However, the average yield of Victorgrain 48-93 in three different tests at Clemson in 1957 was 62 percent lower than Arlington in these same tests. If we may use these comparison data as a basis for measuring the reduction in oat yields due to the presence of this chlorotic disease the results are phenomenal.

SOUTH CAROLINA AGRICULTURAL EXPERIMENT STATION

OAT DISEASES IN MISSISSIPPI[1]

S. S. Ivanoff, Donald H. Bowman, P. G. Rothman[2]

Fungus diseases on oats in Mississippi developed to unprecedented proportions during the 1956-1957 season, as a result of very favorable conditions of temperature, moisture, and relative humidity.

Crown rust (Puccinia coronata) appeared to a very noticeable extent early in the fall of 1956 on all commercial varieties, including Victorgrain 48-93, Delta Red 88, and Nortex 107. In south Mississippi the grain yields were probably cut an average of 25 percent or more by this disease alone. More definite estimates were difficult to make because of the interference of other diseases referred to below. It would be correct, however, to state that 1956-1957 was the worst crown rust year in Mississippi during the last 10-year period.

Victoria blight (Helminthosporium victoriae) was severe in south Mississippi, adding to the damage caused by crown rust in Victoria-derived varieties. This is the first appearance of this disease on a large scale in Mississippi since the epidemics of 1946-1948. What might be a new symptom on the glumes is described later in this article (see "empty hull").

Leaf spot (Helminthosporium avenae) was found to some extent in every field inspected. The actual damage was difficult to estimate because of other leaf spot diseases caused by species of Helminthosporium. The recently reported "black stem" symptom presumably caused by the same pathogen (5) was evident in late maturing oats of all varieties in every county visited, but particularly in Newton, Oktibbeha, and Monroe. The purpling of both stems and leaves was quite in evidence. The spread of this disease requires close watching (2, 3, 5).

Culm rot (strain of Helminthosporium sativum) was identified at several locations.

Smut (Ustilago avenae) was very noticeable, particularly on the Red Rustproof type oats, some fields showing up to 25 percent infection.

Red leaf (virus) was observed up to 20 percent in some fields, which is the highest incidence ever observed for this disease in this State.

"Empty hull" condition (cause unknown). An unusual feature of the 1956-1957 disease situation was the general appearance of a puzzling, and perhaps complex, disease condition. There were many oat fields in south Mississippi which even at close range appeared quite normal -- tall, vigorous, and healthy -- but which yielded only a fraction of the expected grain. Some of these fields produced no grain whatsoever. This was particularly true of Victorgrain 48-93, which was extensively planted. The good appearance of these fields was so deceiving that some growers started combining operations with expectations of a good grain crop. It was quickly apparent, however, that the harvested grain consisted for the most part of hard, well-formed, but empty hulls. This "empty hull" condition of the crop was most prevalent in south Mississippi, less prevalent in central and north Mississippi, and much less so in the Delta region. It was found on all commercial and experimental varieties observed. Upon closer examination it was observed that the color of the upper portion of the panicles was not quite normal. This color may best be described as faintly peach. It is important to note that the roots of most "empty hull" plants appeared normal.

A somewhat similar situation was observed in three isolated fields of Victorgrain 48-93 in 1953 in Newton County in central Mississippi. However, in these fields there was also an excessive dropping of many spikelets containing well filled kernels, in addition to the empty hull

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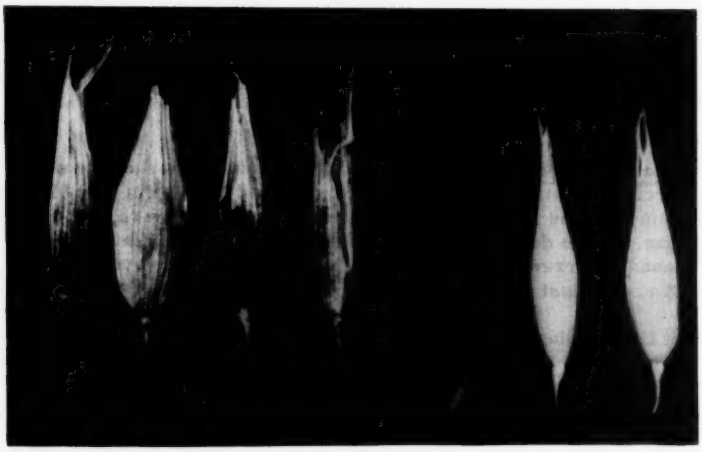

FIGURE 1. Left. Oat kernels of the variety Victorgrain 48-93 showing dark spore masses of Helminthosporium on the glumes. Culture isolates made from this material were tested for pathogenicity in the greenhouse on Victorgrain 48-93, Nortex 107, Delair, and Mid-South oats. Victorgrain 48-93 came down with Victoria blight, and the plants died in a few days. The other varieties were not affected. Right. Healthy kernels for comparison.

condition. The affected panicle branches were dark or purple-black in color. This disease was at that time described and named "spikelet-drop" by the senior author (4). What relationship "spikelet-drop" may have etiologically to "black stem" and to the "empty hull" condition remains to be investigated.

The cause of the "empty hull" condition remains unknown. Conspicuous black spore masses (of a species of Helminthosporium) on the glumes were very often associated with this condition, particularly on the variety Victorgrain 48-93 (Fig. 1). They formed a more or less definite pattern. The individual spores resembled those of H. victoriae but did not seem typical of this species because of their yellowish color and somewhat different shape. Several isolates were secured from the infected glumes and their pathogenicity was tested under humid conditions in the greenhouse on four oat varieties, Victorgrain 48-93, Nortex 107, Delair, and Mid-South. Only the Victorgrain 48-93 plants became infected, dying within a few days. The first symptoms to appear were light brown lesions on the leaves similar to those previously reported to be induced by H. victoriae on mature Victorgrain plants (1).

In conclusion, it may be stated that the combined disease damage to the 1956-1957 oat crop in Mississippi was excessive. Not all diseases, particularly those caused by various species of the genus Helminthosporium, were definitely identified. This seems to be a disease or disease complex not well understood. Further investigations with pure fungus cultures under strict environmental control are needed.

Frequent rains from October through December prevented many growers throughout the State from planting in the fall of 1957. Oats planted in September for grazing are doing well except in places where Victoria blight completely destroyed susceptible varieties. Much less damage, up to 15 percent, was caused by Victoria blight on later plantings of Alamo and Victorgrain 48-93 in Hinds and Claiborne Counties. The limited damage in the later plantings was probably due to lower soil temperatures. It seems likely that this disease may again cause serious damage on susceptible varieties in the Spring of 1958.

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MISSISSIPPI AGRICULTURAL EXPERIMENT STATION IN COOPERATION WITH CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AT STONEVILLE, MISSISSIPPI.

LOOSE SMUT REACTIONS OF SOME WINTER WHEAT INTRODUCTIONS[1]

John F. Schafer, Ralph M. Caldwell, Leroy E. Compton, and Fred L. Patterson[2]

Loose smut caused by Ustilago tritici (Pers.) Rostr. has continued to be a major disease of wheat in Indiana. Strains of the fungus which are pathogenic to wheat varieties possessing resistance derived from Trumbull have become widespread and seriously damaging throughout the State in recent years.

This occurrence of virulent strains on a wheat variety that had been highly resistant over many years emphasizes the hazard that varieties presently resistant may yet be attacked by newer races of the highly variable smut fungus. In view of this potential, it is essential to identify additional unrelated sources of resistance and initiate utilization of these in breeding programs.

A block of 300 winter wheat varieties of foreign origin was grown at Lafayette, Indiana, during 1951-52 as part of the cooperative program conducted by Purdue University and the United States Department of Agriculture for the evaluation of varietal sources for superior small grain characters[3]. A number of these varieties possessed sufficient hardiness to survive the winter and at anthesis in 1952 were inoculated with a composite of Trumbull-attacking cultures of Ustilago tritici. Some entries which were missed the first year were inoculated in one of the following two years, and some of the more promising varieties were reinoculated. Inoculation was accomplished using Cherewick and Popp's modification of Moore's partial vacuum method[4]. Approximately five heads of each variety were inoculated and planted into individual rows. The varieties tested, obtained primarily from Portugal and Spain, and results of these inoculations are listed in Table 1.

Seven varieties, P.I. numbers 191232, 191533, 191570, 191704, 191706, 191733, and 191749, were selected for more intensive testing on the basis of their low loose smut percentages in 1953 following the 1952 inoculation. Although these varieties have continued to exhibit some degree of loose smut resistance, all showed the presence of a limited amount of smut in 1954 following natural infection in the 1953 test nursery. P.I. 191232 was obviously mixed for plant type. The incidence of loose smut in the other varieties is presented in Table 2.

These selected varieties were tested with six cultures of U. tritici chosen for virulence on wheat varieties possessing resistance derived from Trumbull. Each variety has also been reinoculated with its own culture when possible. Results of this testing for 1955, 1956, and 1957 are presented in Table 3. The sources and selection history of the U. tritici cultures used are listed in Table 4.

On the basis of these tests, the most promising varieties are two obtained from Portugal, P.I. 191733 (Richelle) and P.I. 191749 (Rieti). The highest percentage of infection observed was 5 percent for Richelle with culture V_7 in 1955 and 2 percent for Rieti with culture ET_6 also in 1955.

Other varieties not studied as extensively as these seven but appearing to have possible promise on the basis of the reactions listed in Table 1 are P.I. numbers 191140, 191506, 191586, 191685, and 191899.

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[1] Cooperative investigations between Purdue University and Crops Research Division, Agricultural Research Service, United States Department of Agriculture. Journal Paper No. 1227, Purdue University Agricultural Experiment Station.

[2] Associate Professor and Professor of Plant Pathology, Purdue University; Agronomist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture; and Professor of Agronomy, Purdue University.

[3] Seed was supplied by David J. Ward from the World Collections of Small Grains of the Crops Research Division, United States Department of Agriculture.

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Table 1. Percent of loose smut infection of some wheats of foreign origin following inoculation with Ustilago tritici, Lafayette, Indiana

P. I. Number	Name	Country of origin	Average estimated percent of infection (number of heads tested in parentheses) 1953	1954	1955
191140	Mocho	Spain		2(4)	
191176	Navarro 325		43(5)		
191184	Rapen de Asturias		9(5)		
191199	Rojo de Humanes		10(4)		
191199-1	Rojo de Humanes sel.				71(4)
191200	Rojo Sabàndo		24(6)		
191207	Ruiz de Arroniz		25(2)		46(4)
191211	San Rafael		35(4)		
191217	Toseta de Huesca		89(5)		
191219	Toseta de Navascues		13(2)		
191226	Varrone x Argon 03			17(4)	
191229	Vitoria 1		78(4)		
191232	Xexa de Alicante		tr(4)	1(4)	
191233	Xexa del Vall		57(3)		
191234	Yugo-Eslavo		22(5)		
191234-3	Yugo-Eslavo sel.			49(5)	
191240	Jeja de Conesa		8(5)		
191240-1	Jeja de Conesa sel.			14(4)	
191244	Jeja Villar de Domingo Garcia		15(5)		
191245	Jeja de Almendros		15(5)		
191275	Moskowskaia	Russia	34(5)		
191502	Hamisa semi-ariste 436-10099	Portugal			9(6)
191506	Rosafe Argelino 13961			0(3)	5(5)
191513	H 33 E 13402		12(5)		
191513-5	H 33 E 13402 sel.			3(5)	4(3)
191514	H 32 D 13393		65(2)		
191516	H 15 E 13338		52(3)		
191530	HNAD 12231		14(2)		
191530-2	HNAD 12231 sel.			1(3)	
191532	H 15 K 13349		85(2)		
191533	H 39 L 13640		2(5)	4(5)	
191533-5	H 39 L 13640 sel.			5(5)	
191558	H 13 F 13340		15(2)		
191570	H 36 B 12744		4(5)	tr(3)	
191586	H 39 J 13635 A			0(2)	
191591	H 23 C 12724			10(4)	
191663	Strampelli 9378			7(5)	
191685	Touzelle rouge de Provence 2962			0(2)	

Table 1. Concluded.

P. I. Number	Name	Country of origin	Average estimated percent of infection (number of heads tested in parentheses) 1953	1954	1955
191693	Gironde 3550 A	Portugal	10(4)		
191704	Tremezino Ariz 8556		0(4)	2(4)	
191705	Australia 13623		34(5)		
191706	Bladette de Ble Splos		tr(4)		
191723	Warrone 9383		32(5)		
191733	Richelle Blanche Hative 2693 v. 1		tr(6)*	0(2)	
191733-4	Richelle Blanche Hative sel.			0(1)	
191733-5	Richelle Blanche Hative sel.			1(5)	
191733-6	Richelle Blanche Hative sel.			3(5)	
191749	Rieti 2669		0(3)	tr(4)	
191768	HN Rod 36 13783		9(2)		
191899	Arrancada J7 13683			0(2)	
192050	Pombinho 0317		11(5)		
192186	Amarai Tipo 90		43(2)		
192239	Hatvani 5613		56(4)		

* One additional head was 95 percent infected

Table 2. Natural incidence of loose smut infection in six selected foreign wheat varieties in 1954 at Lafayette, Indiana

P. I. Number	Name	Number loose smut infected heads in 10 4-foot rows
191533	--	11
191570	--	6
191704	Tremezino	4
191706	Bladette	30*
191733	Richelle	4
191749	Rieti	4**

* Based on 4 rows
** Based on 8 rows

Table 3. Percent of loose smut infection in seven selected foreign wheat varieties following inoculation with individual cultures of Ustilago tritici at Lafayette, Indiana.

Culture	Year	Varieties tested and average estimated percent of infection (Number of heads tested in parentheses)						
		PI 191232 Alicante	PI 191533	PI 191570	PI 191704 Tremezino	PI 191706 Bladette	PI 191733 Richelle	PI 191749 Rieti
$2E_4$	1955		tr(5)	4 (5)	1 (4)	4 (4)	1 (5)	0 (5)
$2E_5$	1956	8 (9)	1 (4)	0 (2)	0 (5)		0 (4)	0 (4)
$2E_6$	1957	23 (5)		4 (6)	0 (5)	0 (5)	0 (5)	
ET_6	1955		13 (5)	30 (3)	2 (5)	16 (5)	3 (5)	2 (5)
ET_7	1956	0 (4)	2 (5)		0 (5)	tr (5)	tr (4)	0 (4)
ET_8	1957	0 (5)	2 (3)	10 (4)	14 (5)	34 (5)	0 (3)	0 (5)
$AT_2W_1T_{12}$	1955		5 (5)	4 (5)	1 (4)	2 (4)	tr (5)	tr (5)
$AT_2W_1T_{13}$	1956	9 (4)	8 (4)		0 (4)	1 (4)	tr (4)	0 (3)
$AT_2W_1T_{14}$	1957	25 (5)	36 (5)	4 (4)	33 (4)	7 (5)	0 (5)	0 (5)
ThT_{10}	1955		6 (5)	11 (5)	4 (5)	9 (5)	1 (4)	0 (5)
ThT_{11}	1956	0 (3)	4 (5)	3 (4)	0 (2)	0 (5)	0 (5)	0 (4)
ThT_{12}	1957	1 (5)	21 (5)	12 (5)	29 (5)	0 (5)	0 (5)	tr (5)
Th_9Se_1	1955		5 (5)	9 (5)	9 (5)	9 (5)	1 (5)	tr (5)
Th_9Se_2	1956	0 (5)	10 (4)	23 (3)	0 (2)	0 (5)	3 (5)	0 (5)
Th_9Se_3	1957	12 (5)	13 (5)	41 (5)	2 (5)	4 (4)	0 (5)	0 (5)
V_7	1955		8 (4)	7 (5)	1 (5)	12 (5)	5 (5)	tr (4)
V_8	1956	0 (5)	1 (5)	4 (5)	0 (4)	2 (10)	tr (5)	
V_9	1957	4 (5)		4 (4)	2 (5)	0 (5)	0 (4)	0 (5)
Al_1	1956	4 (14)						
Al_2	1957	11 (14)						
Pi3	1955		12(15)					
$Pi3_1$	1956		8(10)					
$Pi3_2$	1957		30(20)					
PiO	1955			6(15)				
Tr	1955				12(15)			
Tr_1	1956				3(11)			
Tr_2	1957				50(20)			
Bl	1955					16(14)		
Bl_1	1956					tr(14)		
Bl_2	1957					2(20)		
Rc	1955						2(15)	
Rc_1	1956						tr (14)	
Rc_2	1957						0(20)	
Re	1955							1(15)

* See Table 4 for source and history of cultures.

Table 4. Source and selection history of cultures of Ustilago tritici used for inoculation.

Culture	Source variety		Year of collection	Subsequent culture history
$2E_4$	Knox & related lines	Lafayette, Ind.	1950	4 passages through Knox
$2E_5$				5 " " "
$2E_6$				6 " " "
ET_6	Knox relative	Lafayette, Ind.	1948	6 passages through Trumbull
ET_7				7 " " "
ET_8				8 " " "
$AT_2W_1T_{12}$	Ashland	Lexington, Ky.	1939	2-Trumbull, 1-Wabash, 12 Trumbull
$AT_2W_1T_{13}$				" " "
$AT_2W_1T_{14}$				" " "
ThT_{10}	Thorne	Lafayette, Ind.	1944	10 passages through Trumbull
ThT_{11}				11 " " "
ThT_{12}				12 " " "
Th_9Se_1	Thorne	Lafayette, Ind.	1944	9-Thorne, 1-Seneca
Th_9Se_2				" 2 "
Th_9Se_3				" 3 "
V_7	Vigo	Lafayette, Ind.	1947	7 passages through Vigo
V_8				8 " " "
V_9				9 " " "
Al_1	Alicante	Lafayette, Ind.	1954	1 passage through Alicante
Al_2				2 " " "
Pi3	P.I. 191533	Lafayette, Ind.	1954	---
$Pi3_1$				1 passage through P.I. 191533
$Pi3_2$				2 " " "
PiO	P.I. 191570	Lafayette, Ind.	1954	---
Tr	Tremezino	Lafayette, Ind.	1954	---
Tr_1				1 passage through Tremezino
Tr_2				2 " " "
Bl	Bladette	Lafayette, Ind.	1954	---
Bl_1				1 passage through Bladette
Bl_2				2 " " "
Rc	Richelle	Lafayette, Ind.	1954	---
Rc_1				1 passage through Richelle
Rc_2				2 " " "
Re	Rieti	Lafayette, Ind.	1954	---

PURDUE UNIVERSITY AGRICULTURAL EXPERIMENT STATION AND CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE.

CO-OPERATIVE SEED TREATMENT TRIALS - 1957[1]

J. E. Machacek[2]

Abstract

Sixteen newly introduced seed dressings were compared in 1957 with three standard ones at 24 stations in Canada and the United States. All were tested in the field against bunt of wheat (mixed Tilletia foetida (Wallr.) Liro and T. caries (DC.) Tul.), loose and covered smut of oats (mixed Ustilago avenae (Pers.) Rostr. and U. kolleri Wille), covered smut of barley (U. hordei (Pers.) Lagerh.), and seed rot of flax resulting from threshing injury. They were also tested in the greenhouse against seedling blight (seed- and soil-borne pathogens) of the three cereals, and for their phytotoxicity to all four crops. The results obtained are presented in tabular form. It is thought that of the field data obtained only those pertaining to barley and flax are acceptable, because very little smut developed in wheat and a great deal of smut developed in oats -- in the latter crop even when the seed was treated with products that are usually effective. The results obtained with oats in 1957 and in the two previous years suggest that the "blendor" method of seed inoculation may not be suitable for use in comparisons of seed dressings.

MATERIALS AND METHODS

In 1957, three standard and 16 new or recently introduced seed dressings were tested against the common seedling-infecting smuts and seedling blights of wheat, oats and barley, and against seed rot of flax. These seed dressings were:

Aldmer -- A wettable powder containing 40.0% aldrin and 2.0% mercury as a mixture of phenyl mercury acetate and ethyl mercury chloride. Obtained from Chipman Chemicals Limited, Winnipeg, Manitoba.

Captan 50 W -- A wettable powder containing 50.0% captan. Obtained from Chipman Chemicals Limited, Winnipeg, Manitoba.

Canuck Liquid Mercury 50 -- A liquid containing 3.8% mercury as phenyl mercury acetate. Obtained from Gallowhur Chemicals Canada Limited, Montreal, Quebec.

Ceresan 75 -- A liquid containing 2.04% mercury as a mixture of ethyl mercury 2,3 dihydroxy propyl mercaptide and ethyl mercury acetate. Obtained from I. E. du Pont de Nemours, Wilmington, Delaware.

Ceresan 100 -- A liquid containing 2.76% mercury as a mixture of ethyl mercury 2,3 dihydroxy propyl mercaptide and ethyl acetate. Obtained from I. E. du Pont de Nemours, Wilmington, Delaware.

Ceresan M -- A wettable powder containing 3.2% mercury as ethyl mercury p-toluene sulfonanilide. Used as a standard of comparison for seed dressings in powder form. Obtained from I. E. du Pont de Nemours, Wilmington, Delaware.

Dual Purpose Bunt-No-More -- A powder containing 40.0% heptachlor and 13.0% hexachlorobenzene. Obtained from Green Cross Insecticides, Montreal, Quebec.

Formaldehyde -- A liquid containing 37.0% formaldehyde. Obtained from Standard Chemical Company Limited, Toronto, Ontario.

Gallotox 50 -- A liquid containing 3.88% mercury as phenyl mercury acetate. Obtained from Gallowhur Chemicals Canada Limited, Montreal, Quebec.

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Gallotox 75 -- A liquid containing 2.98% mercury as phenyl mercury acetate. Obtained from Gallowhur Chemicals Canada Limited, Montreal, Quebec.

Isotox-PMA -- A powder containing 45.0% lindane and 1.0% mercury as phenyl mercury acetate. Obtained from Ortho Agricultural Chemicals Limited, Vancouver, British Columbia.

Metasan M -- A powder containing 3.2% mercury as methyl mercury oxyquinolinate. Obtained from Natural Products Corporation, Montreal, Quebec.

Metasol M -- A liquid containing 1.5% mercury as methyl mercury oxyquinolinate. Obtained from Natural Products Corporation, Montreal, Quebec.

Mer-sol 7 -- A liquid containing 4.0% mercury as phenyl mercury ammonium acetate. Obtained from Stauffer Chemical Company, Omaha, Nebraska.

Mer-sol 51 -- A liquid containing 2.67% mercury as mixed phenyl mercury acetate and ethyl mercury acetate. Obtained from Stauffer Chemical Company, Omaha, Nebraska.

"New" Gallotox -- A liquid containing 3.9% mercury as phenyl mercury acetate. Obtained from Gallowhur Chemicals Canada Limited, Montreal, Quebec.

Panogen 15 -- A liquid containing 1.5% mercury as methyl mercury dicyan diamide. Used as a standard of comparison for seed dressings in liquid form. Obtained from Panogen, Inc., Ringwood, Illinois.

Smut-B-Gon -- A liquid containing 4.0% mercury as phenyl mercury acetate. Obtained from California Spray-Chemical Corporation, Richmond, California.

Thiram 75 -- A wettable powder containing 75% thiram. Obtained from Chipman Chemicals Limited, Winnipeg, Manitoba.

The seed used in these trials was as follows:

Wheat bunt trials -- Variety Thatcher. Seed artificially contaminated (1:200, by weight) with mixed spores of Tilletia foetida and T. caries.

Oat smut trials -- Variety Vanguard. Seed was first artificially contaminated (1:200, by weight) in bulk with mixed spores of Ustilago avenae and U. kolleri, and then given a 10-second hull-loosening treatment in a Knapp-Monarch food homogenizer operating at medium speed. The seed was then dried for a week before any seed dressings were applied to it.

Barley smut trials -- Varieties mixed (farm sample). Very heavy natural contamination by Ustilago hordei.

Flax seed rot trials -- Variety Rocket. About 50 percent of the kernels were cracked during threshing.

In all tests, the seed was held in closed glass jars for at least a day after it was treated. It was then packaged and sent to the co-operators. Each group of envelopes holding seed of the same kind and treated with the same seed dressing was wrapped with cellophane to protect it from the fumigant action of other seed dressings in the same parcel of seed.

EXPERIMENTAL RESULTS

The results obtained from field experiments with treated seed are summarized in Table 1, and those from greenhouse experiments in Table 2. Table 1 shows the degree to which smut was controlled in wheat, oats, and barley, and to which seed rot was controlled in flax. It is thought that the results obtained with barley and flax correctly indicate those obtainable under ordinary farm conditions. The results with wheat and oats, on the other hand, are considered to be unsatisfactory -- the ones for oats because "too much" smut developed in the crop from treated seed, even when the products used for seed treatment previously gave complete control of smut in the oat crop, and the ones for wheat because there was too little smut. It is not known what prevented the development of bunt in wheat, because a pre-inoculation test showed spore germination to be satisfactory and because at some stations sowings under the same conditions of inoculated wheat from other sources produced a heavily smutted crop. The heavy development of smut in oats can be attributed solely to the method (by food homogenizer) of seed inoculation, as the same situation (lack of control of oat smut by formerly very effective treatments) was found in experimental plots during three consecutive summers and in large beds of soil in the greenhouse during two consecutive winters. Tests are under

Table 1. Summary of field data from co-operative seed treatment trials of 1957.

Treatment	Dose (oz.:bu.)				Smut (percent)			Germination (percent)[b]
	Wheat	Oats	Barley	Flax	Wheat	Oats	Barley	Flax
Untreated dry seed	0.0	0.0	0.0	0.0	2.90	47.3	27.2	36.2
Powder								
Ceresan M	0.5	0.5	0.5	1.5	0.65	23.2	1.5	47.2
Aldmer	2.0	1.4	1.4	5.0	0.59	26.0	4.2	43.3
Captan 50W	1.0	1.0	1.0	3.0	1.15	46.9	9.3	58.6
Dual Purpose								
Bunt-no-More	2.0	1.4	1.4	5.0	1.00	52.3	30.7	37.5
Isotox-PMA	2.0	1.4	1.4	5.0	0.90	26.4	8.6	43.0
Metasan M	0.5	0.5	0.5	1.5	0.68	21.6	1.6	47.9
Thiram 75	0.5	0.5	0.5	1.5	0.43	47.4	18.0	53.4
Liquid								
Panogen 15	0.75	0.75	0.75	1.5	0.65	28.7	3.4	45.2
Canuck Liquid								
Mercury 50	0.75	0.75	0.75	1.5	0.62	41.5	5.9	51.9
Ceresan 75	0.75	0.75	0.75	1.5	0.56	30.5	2.6	49.8
Ceresan 100	0.75	0.75	0.75	1.5	0.96	27.0	4.8	53.9
Formaldehyde	10-minute soak in 1:320 solution			0.0	1.11	13.0	11.4	36.8
Gallotox 50	0.75	0.75	0.75	1.5	0.65	36.4	7.6	48.3
Gallotox 75	0.75	0.75	0.75	1.5	0.75	39.4	12.2	46.8
Mer-sol 7	0.75	0.75	0.75	1.5	0.81	39.5	20.9	47.7
Mer-sol 51	0.75	0.75	0.75	1.5	1.21	30.5	6.7	48.6
Metasol M	0.75	0.75	0.75	1.5	0.78	31.0	10.0	51.6
"New" Gallotox	0.75	0.75	0.75	1.5	1.00	37.0	3.9	51.5
Smut-B-Gon	0.5	0.5	0.5	1.5	0.53	40.1	18.7	47.1
Significant difference (5%)					0.14	12.0	3.1	5.4

[a] Means of data from 4, 16, and 18 stations for wheat, oats, and barley, respectively.
[b] Means of data for 11 stations.

way to determine which of several methods of artificial seed inoculation will be a satisfactory substitute for natural contamination. It is not thought necessary to comment further on the data presented in Table 1, as they otherwise seem to be self-explanatory.

Table 2 shows the effect of storage on the germination of treated seed. From the data given, it is evident that in some instances storage of the treated seed in closed glass jars injured seed germination. Wheat seemed to be the most susceptible of the four crops, as it was injured by Aldmer, Isotox-PMA, Metasan M, Canuck Liquid Mercury 50, Ceresan 75, Ceresan 100, Gallotox 50, Mer-sol 51, and "New" Gallotox. Isotox-PMA and formaldehyde, even without storage, injured the seed to a considerable degree. It is possible that the wheat seed used was particularly susceptible to chemical injury although it was of "Registered" stock of high germinability. Isotox-PMA and formaldehyde lowered germination of oats, the injury appearing to be less in the case of formaldehyde at the beginning of storage than later, and the reverse holding for Isotox-PMA. Barley seed treated with formaldehyde also lost some of its germinability in storage. Flax seemed to be quite resistant to the action of most of the chemicals used, as only Mer-sol 51 caused an appreciable drop in germination.

Table 2. Effect of storage on the germination of treated seed. Summary of greenhouse data relating to phytotoxicity of the seed dressings included in the co-operative seed treatment trials of 1957.

Product	Dose (oz.:bu.)				Percent germination after indicated duration of storage (months)[b]							
	Wheat	Oats	Barley	Flax	Wheat		Oats		Barley		Flax	
					0	4	0	4	0	4	0	4
Untreated dry seed	0.0	0.0	0.0	0.0	78.5	83.0	65.0	47.0	55.5	42.5	27.5	27.5
Powder												
Ceresan M	0.5	0.5	0.5	1.5	87.5	60.5	72.5	63.0	61.5	49.5	60.0	55.0
Aldmer	2.0	1.4	1.4	5.0	85.0	11.0	74.0	60.5	56.5	51.5	65.5	56.5
Captan 50W	1.0	1.0	1.0	3.0	87.0	94.0	70.0	66.0	58.0	54.5	75.0	63.5
Dual Purpose Bunt-No-More	2.0	1.4	1.4	5.0	74.5	75.5	56.0	62.5	52.0	36.0	33.5	37.5
Isotox-PMA	2.0	1.4	1.4	5.0	25.5	3.0	21.5	32.5	55.5	44.0	54.5	48.5
Metasan M	0.5	0.5	0.5	1.5	90.5	23.0	67.0	63.5	47.5	47.0	65.5	52.5
Thiram 75	0.5	0.5	0.5	1.5	89.5	87.0	69.0	55.0	62.5	45.0	51.0	52.0
Liquid												
Panogen 15	0.75	0.75	0.75	1.5	88.5	84.5	69.5	61.5	59.0	53.0	49.0	50.0
Canuck Liquid Mercury 50	0.75	0.75	0.75	1.5	88.0	45.5	64.0	64.5	64.0	49.5	63.5	59.4
Ceresan 75	0.75	0.75	0.75	1.5	88.0	9.5	77.5	64.0	65.0	49.5	59.5	62.0
Ceresan 100	0.75	0.75	0.75	1.5	86.5	13.5	75.0	52.0	55.0	45.0	63.0	64.0
Formaldehyde	10-minute soak in 1:320 solution			0.0	1.0	2.0	37.5	19.0	43.5	29.5	39.0	37.0
Gallotox 50	0.75	0.75	0.75	1.5	83.0	33.0	65.5	63.0	50.0	44.0	59.5	63.5
Gallotox 75	0.75	0.75	0.75	1.5	83.0	60.5	69.5	62.0	50.5	40.5	57.5	52.0
Mer-sol 7	0.75	0.75	0.75	1.5	87.0	67.0	75.0	58.5	51.5	41.0	49.0	48.5
Mer-sol 51	0.75	0.75	0.75	1.5	86.5	6.0	70.0	52.0	53.5	47.0	62.0	40.0
Metasol M	0.75	0.75	0.75	1.5	89.0	74.5	76.0	60.0	55.5	41.5	57.5	49.5
"New" Gallotox	0.75	0.75	0.75	1.5	71.0	7.0	75.5	64.0	58.0	50.0	66.5	56.0
Smut-B-Gon	0.5	0.5	0.5	1.5	88.0	56.5	69.0	62.0	63.5	40.5	60.5	62.5
Significant difference (5%)					10.8	17.9	8.7	9.7	9.1	DNS[a]	13.0	12.0

[a] DNS = Differences not significant.

[b] A germination test was made within one week after treatment and again after four months.

The following cooperators helped with the trials of 1957: Dr. M. C. Shurtleff and Mr. A. Kelley (Ames, Iowa); Mr. E. C. Stacey and Mr. A. G. Guitard (Beaverlodge, Alberta); Mr. W. H. Johnston (Brandon, Manitoba); Dr. C. M. Nagel and Dr. J. F. Hennen (Brookings, South Dakota); Mr. J. E. Campbell (Charlottetown, Prince Edward Island); Dr. A. W. Henry and Dr. L. E. Tyner (Edmonton, Alberta); Dr. W. E. Brentzel (Fargo, North Dakota); Mr. P. N. Grainger (Fredericton, New Brunswick); Dr. W. Crosier (Geneva, New York); Dr. S. G. Fushtey (Guelph, Ontario); Mr. R. I. H. McKenzie (Indian Head, Saskatchewan); Mr. M. L. Kaufmann (Lacombe, Alberta); Mr. J. S. Horricks (Lethbridge, Alberta); Dr. D. C. Arny (Madison, Wisconsin); Mr. H. R. Ballantyne (Melfort, Saskatchewan); Mr. W. J. Breakey (Morden, Manitoba); Mr. William Bell (Ottawa, Ontario); Dr. L. H. Purdy (Pullman, Washington); Dr. R. O. Lachance (Ste. Anne de la Pocatiere, Quebec); Mr. M. B. Moore (St. Paul, Minnesota); Prof. T. C. Vanterpool and Dr. R. C. Russell (Saskatoon, Saskatchewan); Mr. A. G. Kusch (Scott, Saskatchewan); Mr. D. S. McBean (Swift Current, Saskatchewan); Mr. H. A. H. Wallace (Winnipeg, Manitoba).

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PUCCINIA POAE-NEMORALIS OTTH ON PHLEUM PRATENSE L. AND OTHER GRASS HOSTS IN WYOMING

W. G. Solheim[2]

In the summer of 1957 the author made two collections of rust on Phleum pratense L. Both collections were made in the same general locality within 2 miles of each other in the Grand Teton National Park in the vicinity of Swan Lake. The two specimens, with other rust materials, were sent to Dr. George B. Cummins for identification. He reported the rust on timothy to be Puccinia poae-nemoralis Otth II (= P. poae-sudeticae (Westend.) Joerst. of Arthur's Manual).

The two collections were made at an interval of over a month, the first one on July 27 and the second on September 2. It is interesting to note that Stage III did not develop during this interval. Arthur, in his Manual (p. 151), states that "telia are sparingly produced." He further states that telia have been recorded as occurring in Wyoming (and elsewhere) on certain species of Poa.

This report appears to be the first record of this rust on Phleum pratense. A single specimen of the rust, collected by E. Bethel at Tolland, Colorado, and filed in the National Fungus Collections, is listed as occurring on P. ?pratense. Fortunately the specimen contains a spike of the host plant. Dr. F. J. Hermann examined the specimen and determined the host as P. alpinum L. Dr. Cummins states that the Arthur Herbarium contains four Bethel numbers on this host from Colorado. The rust has also been reported from Alaska on P. alpinum.

Since the rust does parasitize P. pratense, it is strange that it has not previously been reported on this host. The host and rust are both common throughout Wyoming, as well as in other Rocky Mountain States. The known host range for Wyoming is as follows:

GRAMINEAE:

Tribe Agrostideae -- Agrostis alba L., Phleum pratense L.

Tribe Aveneae -- Trisetum spicatum (L.) Richt.

Tribe Festuceae -- Catabrosa aquatica (L.) Beauv., Poa spp., P. alpina L., P. arida Vasey (syn. P. pratensiformis Rydb.), P. epilis Scribn., P. interior Rydb., P. leptocoma Trin., P. nervosa (Hook.) Vasey (syn. P. wheeleri Vasey), P. palustris L., P. pratensis L.

UNIVERSITY OF WYOMING

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[1] Contribution from the Department of Botany and the Rocky Mountain Herbarium of the University of Wyoming, No. 233.

[2] Professor of Botany, University of Wyoming. The author gratefully acknowledges a Grant-in-Aid from the New York Zoological Society for work at the Jackson Hole Biological Research Station of the New York Zoological Society and the University of Wyoming. This Station is located in the Grand Teton National Park near Moran, Wyoming. The author, on sabbatical leave working in the National Fungus Collections, further acknowledges the many courtesies of John A. Stevenson and Paul R. Miller and their staffs.

BOTRYTIS HYACINTHI IN THE UNITED STATES

Charles J. Gould, Maksis Eglitis, and Neil A. MacLean[1]

According to the Plant Disease Survey check list (2), Botrytis hyacinthi Westerd. & v. Beyma has never been reported in the United States. The absence of this pathogen has been interesting in view of the widespread planting of hyacinth bulbs imported from Holland where B. hyacinthi has long been present and serious (1). Its absence in western Washington has been particularly striking because most species of Botrytis thrive under the cool moist climatic conditions of that area. The eventual discovery of B. hyacinthi in Washington was, therefore, more or less to be expected.

The fungus was found first on April 13, 1949, at Mt. Vernon, Washington, growing on the distorted tips of leaves of two plants of the hyacinth variety Pink Pearl. The original bulbs had been imported in the fall of 1948 from Holland. Numerous conidia fitting the description of B. hyacinthi were present on the leaves (Fig. 1) and typical sclerotia were later found on a petiole (Fig. 2). The infected leaf tips were dark brown to almost black, and shrivelled. Very few individual lesions were present.

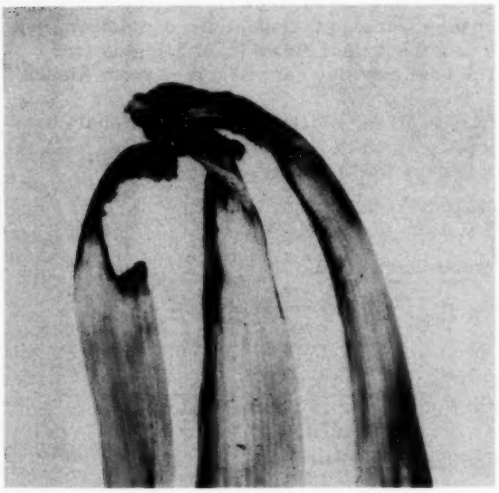

FIGURE 1. Leaf symptoms on hyacinth var. Pink Pearl caused by Botrytis hyacinthi.

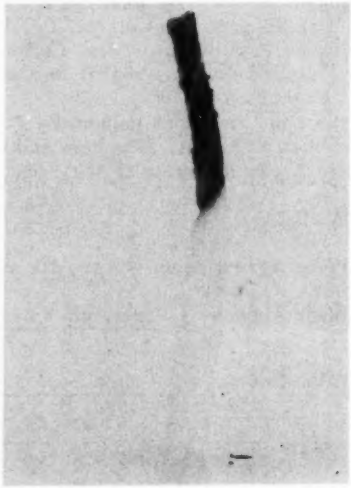

FIGURE 2. Sclerotia of Botrytis hyacinthi on petiole of hyacinth var. Pink Pearl.

The fungus was again collected in Washington, this time near Puyallup, on April 15, 1956. It was found on the varieties Pharos and General Eisenhower grown from bulbs imported the previous fall from Holland. Isolations yielded Botrytis hyacinthi, with a conidial size on malt agar of 12.0-23.5 x 8.3-22.0 microns (average of 100 spores = 17.3 x 11.4 microns), and black sclerotia (average of 50) measuring 1.0-1.7 x 1.0-2.3 mm. Moore (1) lists a range in conidia of 13-19 x 10-14 microns with an average of 16.8 x 12 microns, but does not give any measurements for the sclerotia.

Inoculations with three isolates on hyacinths in a greenhouse gave the following results: 1) Both injured and non-injured unopened flower buds were completely killed and covered with conidia within 12 days after inoculation with conidia, mycelium, or sclerotia; 2) Non-injured leaves remained free from infection, but those injured by burning with a hot needle became

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infected and covered with a mass of conidia when sprayed with a conidial suspension; 3) Infection was not obtained on iris leaves nor on bulbs of iris or daffodils; 4) Twenty-four reisolations from infected flowers and leaves of hyacinths yielded cultures of B. hyacinthi with spore sizes (17.7 x 12.6 microns) comparable with those of the original isolates.

Fortunately, Botrytis hyacinthi apparently did not spread from the original infected plants in either 1949 or 1956, despite the fact that climatic conditions were suitable for the spread of other Botrytis species, such as B. tulipae on tulips. Nevertheless, the appearance of B. hyacinthi is disquieting. The commercial acreage devoted to hyacinths in western Washington is still small, but it has been gradually increasing during the past few years. Since climatic conditions are so favorable for Botrytis development in this area, the establishment of B. hyacinthi, should it occur, would undoubtedly be a retarding factor in the production of hyacinth bulbs in western Washington.

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2. WEISS, FREEMAN, and MURIEL J. O'BRIEN. 1952. Index of Plant Diseases in the United States. Plant Disease Survey, U.S.D.A., Part IV, p. 688.

WESTERN WASHINGTON EXPERIMENT STATION (STATE COLLEGE OF WASHINGTON), PUYALLUP, WASHINGTON AND UNIVERSITY OF BRITISH COLUMBIA, VANCOUVER, CANADA.

ADDITIONAL FINDINGS AS TO THE EFFECTS OF SEVERAL BIOCIDES ON GROWTH OF SEEDLING PINES AND INCIDENCE OF MYCORRHIZAE IN FIELD PLOTS

John G. Palmer and Edward Hacskaylo[1]

In 1957 we reported the effects of several biocides on growth of seedling pines and incidence of mycorrhizae in isolated field plots after one growing season[2]. Pretreatment of the soil with methyl bromide at the rate of 1 pound per 25 square feet prevented formation of mycorrhizae on Virginia and slash pines (Pinus virginiana and P. caribaea). It also increased growth of the seedlings as compared with that of the controls. Vapam and Bedrench were partially effective in preventing development of mycorrhizae. Ethylene dibromide and Nemagon did not decrease development of mycorrhizae, but use of the latter caused stunting of the seedlings. This paper reports the results of examination of the seedlings that remained in the plots (about half) for the second growing season, a total of 18 months.

METHODS

In November 1957, the seedlings were dug and the shoots were separated from the roots. The lengths of the shoots from the root collar to the tip of the leader were measured. The shoots were then dried for 4 days in a forced-draft oven at 80° C and weighed. The extensive root growth made recovery of intact root systems impossible, but observations on the development of mycorrhizae were made.

Table 1. Average heights and dry weights of slash and Virginia pine seedlings grown in chemically treated and control plots for 18 months.

Species of pine and chemical	Number of seedlings	Average shoot height (cm)	Average dry weight of shoot (gm)
Slash pine:			
Methyl bromide	78	52.2	31.1
Ethylene dibromide	67	42.2	21.3
Vapam	73	43.3	21.4
Bedrench	69	43.4	21.0
Nemagon	30	39.8	21.0
None (control)	70	47.9	26.5
L.S.D. (.05)		3.8	5.8
Virginia pine:			
Methyl bromide	40	39.4	27.8
Ethylene dibromide	60	33.4	24.0
Vapam	77	36.5	23.1
Bedrench	77	34.2	20.7
Nemagon	32	24.5	14.5
None (control)	56	32.8	25.8
L.S.D. (.05)		N.S.	6.1

[1]Pathologist and Physiologist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

[2]Hacskaylo, E. and J. G. Palmer, 1957. Effects of several biocides on growth of seedling pines and incidence of mycorrhizae in field plots. Plant Dis. Reptr. 41: 354-358.

RESULTS AND DISCUSSION

Seedlings grown in the methyl bromide plots were tallest and had the heaviest shoots of all, and those grown in the Nemagon plots were shortest (Table 1). The greater growth of the seedlings in plots treated with methyl bromide[2] was partially maintained during the second growing season. The suppression of growth by Nemagon likewise was evident. The initial increased growth of the pines in plots treated with Vapam and Bedrench was not maintained during the second growing season. The Virginia pines were much more branched and irregular in shoot growth than the slash pines. The irregular growth was accentuated by damage of leaders by insects which probably caused the variability in average heights.

Heavy mycorrhizal formations were found on all seedlings including those grown in plots treated with methyl bromide from which mycorrhizae were absent in the first growing season. Possibly residual mycelium or spores in the soil, air-borne spores of mycorrhizal fungi, or a combination of these served as inoculum for the plots. If so, inoculations with specific mycorrhiza-forming fungi would have to be made during the first growing season if methyl bromide were used to eliminate the fungi in the field as a part of controlled pure-culture inoculation experiments.

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TRANSMISSION OF CERATOCYSTIS FAGACEARUM BY INSECTS AND MITES

E. B. Himelick and E. A. Curl[1]

Summary

Results of transmission experiments conducted under natural forest conditions support the theory that certain sap-feeding insects are vectors of the oak wilt fungus, Ceratocystis fagacearum (Bretz) Hunt. Three hundred sixty-nine insects were caught in baited traps over wounds on 92 of 235 oak trees located near sources of oak wilt inoculum in the forest. Three of the 92 trees became infected and wilted. Species of the Nitidulidae were found in each of the traps on the infected trees.

In a greenhouse experiment, the flatheaded borer, Chrysobothris femorata Oliv., transmitted the fungus through feeding wounds which the insects inflicted upon 13 of 54 oak seedlings. In another greenhouse experiment, mites, Garmania bulbicola Oudemans, transmitted the fungus to mechanically made wounds on 11 of 32 oak seedlings.

INTRODUCTION

Above-ground transmission of the oak wilt fungus, Ceratocystis fagacearum (Bretz) Hunt, has received much attention during the past 5 years. Successful experimental transmission of C. fagacearum to wounded oaks by species of the insect family Nitidulidae has been reported by workers in Iowa (13), West Virginia (6, 10), Illinois (9), Wisconsin (11), and Pennsylvania (17). Other workers have demonstrated that the sap-feeding Nitidulidae can carry viable spores of the oak wilt fungus on and in their bodies (1, 15, 16, 18). Further work has revealed that these insects can, under certain experimental conditions, transmit the fungus to fresh wounds on healthy trees (4, 14). Radioactive tracer studies have proved that the sap-feeding Nitidulidae that feed on the oak wilt fungus mats are attracted to fresh wounds on healthy oaks (12).

Griswold et al. (8), Buchanan (2, 3), and Stambaugh et al. (15) reported that bark beetles emerge from diseased trees and carry viable spores of the oak wilt fungus on their bodies. However, transmission of the fungus by beetles through wounds made by the insect vectors has not been reported. Transmission by Drosophila melanogaster (7) and by Collembola (5) has been demonstrated.

The work reported here consisted of two parts. In the first part, baited traps were used to attract insects to wounds made on healthy oak trees growing in the forest. In the second part, transmission tests were conducted in the greenhouse with three species of beetles and a species of mite.

MATERIALS AND METHODS

Forest Experiments

In April, May, and June of 1954 and 1955, experiments were conducted in Sinnissippi Forest in northern Illinois. All of the trees used were either red oak, Quercus borealis var. maxima (Marsh.) Ashe, or black oak, Quercus velutina Lam. They were at least 100 feet distant from oak wilt-affected trees and at least 100 feet from any dead oaks killed by unknown causes. Some of the work was done in black oak reproduction areas where the trees ranged from 1 to 5 inches d. b. h. and where no oak wilt was known to have occurred previously. Fresh fungus mats with the bark and wood intact were removed with a wood chisel from infected trees located in other areas of the forest, and these were transported in plastic bags to the oak reproduction areas. Before removal of the mats, the bark was made fast to the wood with small nails. Thus, any insects that were present on the fungus were transported along with the mats. In areas other than the reproduction area, diseased trees with the fungus

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mats left intact served as sources of inoculum. A few mats having both conidia and ascospores were always present in all the areas where the work was carried out.

Two types of insect traps were used in the forest experiments. One type was designed for trees that measured 6 inches d. b. h. or larger. A hole, 1/2 inch in diameter and 1 1/2 inches in depth, was made in the trunk with a brace and sterilized bit. For bait, mashed ripe banana diluted with water was injected by means of a veterinarian syringe to the back part of the hole, and a piece of 60-mesh brass screen wire was fitted into the hole to prevent the insects from contacting the bait. The rough outer bark around the hole was made smooth with a sterile chisel. Then a cone (Fig. 1, A) of brass screen wire was inserted in the hole, and the protruding flange was stapled to the bark. The cone was 3/4 inch long, 7/16 inch in diameter at the outer end and tapered to a 3/16 inch opening. Prior to insertion of the cone in the hole, a small flap of light weight nylon cloth was fastened to the small end of the cone. The flap was sufficiently flexible to keep the opening of the cone covered and yet allow entry of small insects. In preliminary laboratory trials, this cone type of trap effectively prevented even very active insects, such as fruit flies, from escaping.

The second type of insect trap (Fig. 1, B) was designed for trunks of trees that measured 1 to 5 inches d. b. h. This trap was constructed in three parts. The main body was a stiff plastic cylinder 1 1/2 inches in diameter and 2 inches long. A brass-screen cone with a 1/8-inch opening at the small end was inserted in one end of the cylinder and made fast with small staples and electrician's plastic tape. A more flexible piece of plastic, which was stapled to the opposite end of the cylinder, afforded a means of fastening the trap to a tree. To attract insects to the trap, a vial containing pulverized banana was attached to the bottom of the trap in a vertical position. Attachment of the vial to the trap was made possible by a cork, with a screen-covered hole, previously glued to the plastic cylinder. The hollow cork arrangement made it possible to supply fresh bait when needed. A slanting cut was made in the trunk of each tree with a 1/2-inch sterile chisel, and the insect trap was attached to the tree over the wound. The flexible plastic was wrapped around the trunk, and rubber grafting bands were used to hold the trap securely in place.

Plastic cylinder traps as described above were placed on 205 red oaks and black oaks. Brass screen cone traps were inserted in holes bored in the trunks of 30 trees that measured 6 to 20 inches d. b. h. Closed traps were placed over wounds of 40 additional trees which served as controls. All insect traps, except the controls, were left on the trees for one week, after which the trapped insects were collected and identified[2]. After each trap was removed the wound was protected with grafting wax and tape for the remainder of the experiment.

Greenhouse Experiments

In one of the greenhouse transmission experiments, Chrysobothris femorata Oliv., Agrilus bilineatus Web., and Monarthrum fasciatum Say were enclosed in plastic cages with 3-year-old red oaks (Fig. 1, C, D). These insects had been collected periodically from 2-1/2-foot trunk sections of wilt-killed oaks confined in emergence cans. The insects were placed on 2-week old cultures of the oak wilt fungus 30 minutes before they were transferred to the plastic cages. One or two insects were caged with each tree for a period of 24 hours. Two types of inoculum were used, conidia alone and conidia and ascospores. Seventy-three trees were caged with infested insects, and 20 trees were caged with non-infested insects.

Mites have been observed to be abundant on naturally occurring mycelial mats in Illinois. Also, they have been found occasionally on bodies of bark bores (Fig. 1, E) and various other flying insects. These observations prompted the authors to investigate the possibility of transmission of the oak wilt fungus by mites.

Mites, Garmania bulbicola Oudemans, which were collected from mats on wilt-killed trees, were used in this experiment (Fig. 1, F). The mites were allowed to multiply on 1- to 3-week-old cultures of the oak wilt fungus in petri dishes. Some of the cultures contained conidia only, while others contained both conidia and ascospores. Mites that were to be used in control cages were reared in the following manner. A few mites from one of the fungus cultures were transferred to a petri dish culture of a bacterium which was known to be inhibitory to germination of spores of the oak wilt fungus. After 2 weeks, a few mite eggs were transferred to a culture of Aspergillus sp. Large desiccators, with vaseline-sealed lids, were used as rearing chambers to prevent the mites from migrating from one inoculum to

[2]Insect identifications were made by Dr. Milton W. Sanderson, Taxonomist, Section of Faunistic Surveys and Insect Identification, Illinois Natural History Survey, Urbana, Illinois.

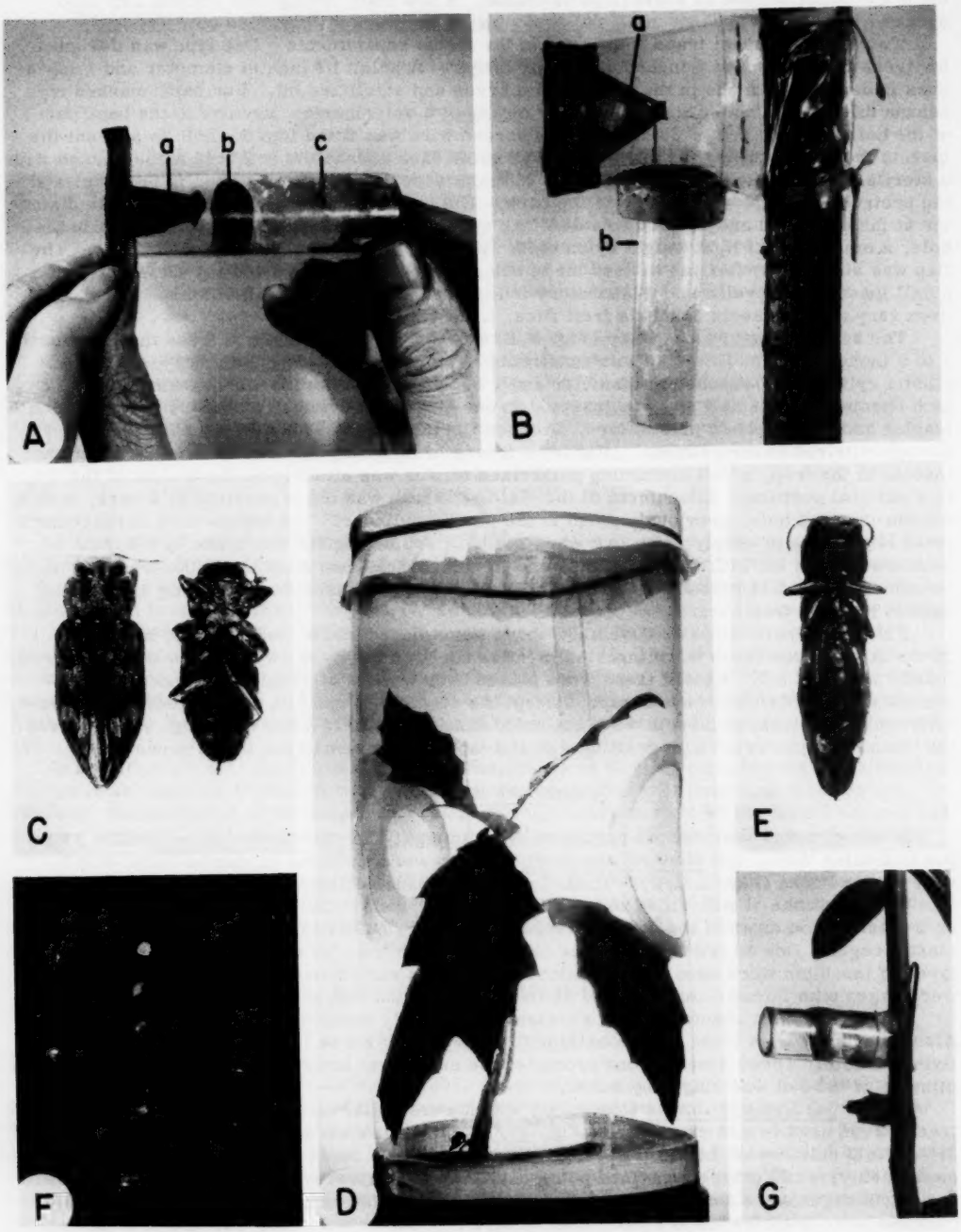

FIGURE 1. Materials and methods used in transmission experiments. A) Brass screen insect trap used on large oaks in forest experiments. The glass vial is not part of the trap, but is used here to represent a bored hole in a tree trunk. a) brass screen cone, b) screen plug, c) banana bait. B) Plastic cylinder trap used on small trees in forest experiments.
(continued on opposite page)

another. Successive generations of mites reared on fresh Aspergillus cultures were used in the controls for the transmission experiment.

The cages used in this experiment consisted of two separate parts. One part was a Van Tieghem cell attached by a plastic sleeve (Fig. 1, G) over the wound at the base of each oak seedling. The wound was a 1/8-inch cut made with a sterile scalpel. All openings around the plastic sleeve were sealed with melted paraffin to prevent escape of the mites. The second part of the cage was a Van Tieghem cell sealed on both ends with a thin film of paraffin to form a small chamber. The mites were introduced into this sealed chamber through a small needle hole in the paraffin. Transfer of the mites from the fungus cultures to the chamber was accomplished with a transfer stick consisting of a paint brush bristle in the end of a 1/8-inch piece of doweling. Four mites were placed in each Van Tieghem cell. After intervals of 15, 30, 60, and 120 minutes, chambers with mites inside were attached to Van Tieghem cells previously fastened to 32 oak seedlings, Quercus virginiana Mill. Melted paraffin was used to seal the two Van Tieghem cells together on each tree. A warm needle was then passed through the paraffin to pierce the inner wall of the Van Tieghem cell that contained the mites, thus allowing the mites to pass into the cell that enclosed the wound. The outer wall of the Van Tieghem cell was sealed with warm paraffin. Intervals of from 15 minutes to 24 hours elapsed between the time wounds were inflicted and the time mites were caged on the wounds. Controls consisted of eight trees on which mites reared for this purpose were caged 15 minutes after wounds were made.

RESULTS

Forest Experiments

Insects were trapped on 73 of 208 trees (35 percent) in 1954, and on 19 of 27 trees (70 percent) in 1955. Thus, insects were trapped on a total of 92 of 235 trees during the 2 years. Five of the species of insects trapped were species of the Nitidulidae (Table 1).

Two of the 73 trees on which insects were trapped in 1954 and one of the 19 trees in 1955 became infected with oak wilt. The insects that were trapped on these three trees are listed in Table 2. The first tree (S-48) that wilted in 1954 had the brass screen type of trap in the trunk. The known source of inoculum nearest to this tree was sporulating mats of the oak wilt fungus on a dead oak tree 140 feet away. The inoculum nearest to tree S-155, which also wilted in 1954, was Nitidulid-infested mats that were placed on the ground 30 feet from the tree. Tree I-12, which wilted in 1955, was 300 feet from the nearest mat-bearing wilt-killed tree. The plastic cylinder type of insect trap was on the trunk of tree S-155 and a brass screen trap was in tree I-12. Eleven insects representing four species of Nitidulidae were found in traps on the three infected trees. Laboratory confirmation of the disease was made in each case. None of the control trees became infected during the period 1954 through 1957.

Greenhouse Experiments

Thirteen of the 54 trees caged with Chrysobothris femorata became infected with the oak wilt fungus and died. The fungus was isolated from all of the infected trees. As far as could be determined, the trees had no wounds other than those made by the insects. Consequently, infection of these 13 trees was assumed to have occurred through wounds made by the insects. Feeding wounds were found at the base of succulent branches on seven of the infected trees. Five other infected trees had feeding wounds at the base of branches, on buds, and on leaves. One of the infected trees, on which a single Chrysobothris beetle had been caged, had a feeding wound on a large vein of one leaf. Nineteen trees caged with Agrilus bilineatus and Monarthrum fasciatum did not become infected. Feeding on the foliage by the Agrilus beetles was only slight, and the Monarthrum beetles did not feed on the trees at any time.

Figure 1 (concluded) -- a) screen cone inserted in cylinder, b) vial with bait attached by cork over screen-covered hole. C) Chrysobothris femorata used in greenhouse experiment. D) Plastic cage used to cage C. femorata on potted oak trees. E) Agrilus bilineatus, recently emerged from wilt-killed oak, with mites attached to its body. F) Mites (Garmania bulbicola) found on mycelial mats of Ceratocystis fagacearum and used in greenhouse experiment. a) Two mite eggs are indicated (15X). G) Mite cage (2 Van Tieghem cells and a plastic sleeve), fastened to a wounded stem of a young live oak.

Table 1. Insects that were caught in two types of traps during field transmission experiments.

Insects	Number of insects caught in each trap			Totals
	Plastic cylinder trap 1954	Brass screen trap 1954	Brass screen trap 1955	
Colopterus truncatus Rand	96	19	20	135
C. semitectus Say	8	16	68	92
Glischrochilus sanguinolentus Oliv.	54	9	7	70
G. quadrisignatus Say	9	1	—	10
Carpophilus niger Say	1	3	1	5
Drosophila spp.	4	1	0	5
Unidentified insects	27	1	24	52
Totals	199	50	120	369

Table 2. Species of insects found in traps on oaks that became infected with the oak wilt fungus.

Tree infected	Size of tree (inches d.b.h.)	Period during which trap was on tree	Date symptoms appeared	Number of insects in traps	Insects found in traps
S-48	7	April 30 to May 7, 1954	July 18, 1954	2	Colopterus truncatus Rand
S-155	2	June 10 to June 17, 1954	August 10, 1954	3	Colopterus truncatus Rand
				2	Colopterus semitectus Say
I-12	2	May 13 to May 20, 1955	September 9, 1955	2	Colopterus truncatus Rand
				1	Colopterus semitectus Say
				1	Carpophilus niger Say

Table 3. Experimental transmission of Ceratocystis fagacearum by mites which were taken from cultures of the fungus and held for 4 time intervals then caged on oak seedlings over wounds of different ages.

Age of wound (hours)	15-minute interval		30-minute interval		1-hour interval		2-hour interval	
	Conidia[a]	Conidia[b] and ascospores	Conidia	Conidia and ascospores	Conidia	Conidia and ascospores	Conidia	Conidia and ascospores
1/4	+	+	−	−			−	−
1/2	−	+			−	−		
1			+	+			−	−
4	+	+			+	−		
8			−	−			−	−
12	+	+			−	−		
24	−	−	−	+			−	−

[a]Mites had fed on cultures of C. fagacearum which had conidia only.
[b]Mites had fed on cultures which had both conidia and ascospores.
+Indicates typical symptoms and positive diagnosis of infection.
−Indicates no wilt symptoms.

Eleven of the 32 trees which were caged with mites in the greenhouse became infected with the oak wilt fungus (Table 3). Five of the infected trees were caged with mites that had been in contact with conidia only and six with mites that had been in contact with both conidia and ascospores. Infection was confirmed by isolating the oak wilt fungus from each tree after wilt symptoms occurred. Infection occurred through wounds that were made on the oak seedlings from 15 minutes to 24 hours prior to the time they were caged with spore-carrying mites. None of the control trees had symptoms of the disease.

DISCUSSION

The results of the insect transmission experiments conducted under natural forest conditions serve as strong evidence that sap-feeding beetles of the family Nitidulidae are important vectors in the spread of the oak wilt fungus. A total of 369 insects was caught in traps over wounds on 92 of 235 trees which were involved in the forest experiments. Since an attractive bait was used to lure the insects to the trees, it cannot be assumed that insects in the forest visit natural wounds with this frequency. However, it has been well established that the Nitidulidae are readily attracted to fresh wounds and that many types of wounds are constantly present on forest trees.

Only three of the 92 experimental trees, on which insects were caught, contracted the disease. This figure is small, but takes on greater significance in view of the fact that the total number of trees used in the experiments was relatively small compared to the number of oaks in the entire forest. Initial infection of three trees in every 235 trees in a forest could cause considerable damage through subsequent spread of the fungus through root grafts.

Chrysobothris femorata, commonly known as the flatheaded apple tree borer, has been found often associated with wilt-killed oaks in nature. The insect breeds in dead or dying oaks, and the larvae make galleries in the cambial zone. Thus, it is possible for the adults to become infested with the oak wilt fungus when they emerge. Observations made in the forest and in the greenhouse indicate that some adults emerge early in the spring and feed on young

succulent twigs and leaves of oak trees. Others that emerged later in the spring were observed to feed on oak leaves until the leaves were about half grown. In the light of these observations and results of the greenhouse experiment, it seems that this insect should be added to the list of possible vectors of the oak wilt fungus.

The possible role of mites in transmitting the fungus cannot be ignored. They occur commonly on mycelial mats of the fungus and occasionally on the bodies of flying insects. The results of the greenhouse experiment reported in this paper demonstrated that mites can carry inoculum on their bodies to wounds on young oak trees, resulting in infection and death of the trees. The failure of mites to transmit the fungus after they were taken from the inoculum source and held in a dry container for 1 hour suggests that the mites died of desiccation or lost the spores that they were carrying, or that the spores lost viability. Nevertheless, a period ranging from 15 minutes to 1 hour would be sufficient time for spore-infested mites to be carried by a flying insect to healthy oak trees in the vicinity of wilted trees. There, the mite, seeking a moist dark place, could enter small wounds and crevices that would not admit large insects.

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SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS NATURAL HISTORY SURVEY, URBANA, ILLINOIS.

THE SMALL OAK BARKBEETLE TRANSMITS THE OAK WILT DISEASE UNDER CAGED CONDITIONS[1]

W. B. Buchanan[2]

The small oak barkbeetle, Pseudopityophthorus minutissimus (Zimm.), has transmitted the oak wilt disease in tests in a nursery at New Franklin, Howard County, Missouri. These tests involved the caging of more than 150,000 beetles on 241 red, pin, and black oak seedlings 3 to 8 feet tall. The tests and results were as follows:

1. a. Bolts, containing about 120,000 beetles, from naturally infected trees were placed in cages on 117 seedlings.
   Results — Oak wilt developed in one seedling, and the fungus, Ceratocystis fagacearum (Bretz) Hunt, was isolated.[3]
   b. Approximately 20,000 beetles reared in the laboratory from similar bolts were placed in cages on 87 seedlings.
   Results -- Oak wilt symptoms developed in one seedling and the fungus was isolated.

2. a. Bolts, containing about 400 beetles, that had been impregnated with a suspension of C. fagacearum under vacuum were placed in cages on six seedlings.
   Results -- No oak wilt symptoms developed.
   b. Approximately 1700 beetles reared from similar bolts were placed in cages on 13 seedlings.
   Results -- Oak wilt symptoms developed in one seedling, but the fungus could not be isolated.

3. a. Bolts, containing about 8000 beetles, from non-diseased trees were placed in cages on ten seedlings.
   Results -- No oak wilt symptoms developed.
   b. Approximately 1000 beetles reared from similar non-diseased bolts were placed in cages on eight seedlings.
   Results -- No oak wilt symptoms developed.

In all of the above tests the beetles fed extensively in twig crotches, bases of leaf petioles, and in rough spots on the stems and larger branches. Although the number of feeding injuries per unit of plant area was greater than usually encountered under natural conditions, the total number of injuries per tree may be far greater on larger trees growing near large quantities of infested material.

The successful transmission of oak wilt by Pseudopityophthorus minutissimus in these

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[1] A more detailed report on this work is being submitted for publication in the JOURNAL OF FORESTRY.

[2] Forest Entomologist, Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, Columbia, Missouri.

[3] All cultures in this study were made by Dr. T. W. Bretz, Professor of Forestry (Pathologist), School of Forestry, University of Missouri, Columbia, Missouri.

Contribution from the Central States Forest Experiment Station, Forest Service, United States Department of Agriculture and Missouri Agricultural Experiment Station, Journal Series No. 1844. Approved by Director.

tests indicates that this beetle may be involved in the overland spread of oak wilt. However, it is apparent from these tests and from field observations that the number that actually spread the disease is very small, otherwise oak wilt infections would be far more common.

CENTRAL STATES FOREST EXPERIMENT STATION, FOREST SERVICE,
UNITED STATES DEPARTMENT OF AGRICULTURE, AND MISSOURI
AGRICULTURAL EXPERIMENT STATION

RESULTS OF ROOT INOCULATIONS WITH THE OAK WILT FUNGUS IN PENNSYLVANIA

W. L. Yount[1]

Summary

Red oak trees in four series, 1954, 1955, 1956, and 1957, were inoculated with Ceratocystis fagacearum in the roots at varying distances from the trunks. Trunk-inoculated trees served as checks. The length of time between inoculation and the first foliage symptoms was longer for the root-inoculated trees in every series. Incubation periods for both trunk- and root-inoculated trees varied extensively from season to season. A majority of the infections from root-inoculated trees in each series developed symptoms at a common date. One or two infected trees from root inoculation in each series did not develop symptoms for a year or more. Inoculation point distance from the trunk was not a significant factor in length of incubation periods.

HISTORY

Jeffery (3) made a study of the time intervals between deaths of trees in 85 oak wilt sites where root grafting seemed to be the means of disease spread. The time intervals varied from within the same year, to 1 year and as long as 6 years. This study suggested that the movement of the pathogen Ceratocystis fagacearum (Bretz) Hunt through the roots from one tree to another is rather slow.

Henry and Riker (2) made inoculation site studies on red and black oaks in Wisconsin. Their report suggests that the incubation period does not vary greatly between root-inoculated and breast height-inoculated trees: root, 21 to 48 days; breast height, 19 to 50 days. Three of eight trees root-inoculated and 46 of 123 inoculated at breast height became infected. No record of individual trees was given (2).

PRESENT WORK

Methods

To test the hypothesis that the movement of the fungus is slow through the roots, ten or more isolated red oak trees were inoculated in the roots in each year from 1954 through 1957, at varying distances from the trunks. It was thought that distance from the trunk would be the major factor in determining how soon foliage symptoms would develop. Trees inoculated farther out on the root would show symptoms last, because the fungus would have to travel farther to reach the stem. In each series, trees inoculated in the trunks at the same time served as checks for comparison[2].

Mycelium and spores scraped from cultures on malt agar served as inoculum for the 1954 and 1955 inoculations. The wound was made with a brace and bit. The 1956 and 1957 trees were wounded with an increment hammer, and the inoculum was a spore suspension in distilled water.

Results

In each year's series of inoculations the incubation periods were longer for root-inoculated trees than for trunk-inoculated trees.

The data do not show a relationship between duration of incubation and inoculation point

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[1] Pathologist, Pennsylvania Department of Agriculture, Harrisburg, Penna. Assistance and advice in this project was supplied by C. L. Morris, Virginia Department of Forestry; W. J. Stambaugh, Pennsylvania State University; J. C. Nelson, Pennsylvania Department of Forests and Waters; Ralph C. Sanderson, Pennsylvania Department of Agriculture; Hugh E. Thompson, Kansas State University.

[2] The check trees (trunk inoculated in Tables 1, 2 and 3) were a part of other research work being done by W. J. Stambaugh.

Tables 1 to 4. Results of root inoculation of red oak trees with the oak wilt fungus, 1954-1957.

Tree No.	Root diameter at inoculation point (inches)	Distance from trunk (feet)	Time to foliage symptoms after inoculation (days)	Crown class
Table 1. Root Inoculations, October 7, 1954.				
1	1	6		
2	1 1/2	3		
3	1 1/2	8		
4	1	4		
5	1 1/2	4	547	
6	1	4		
7	1 1/2	4		
8	1	4		
9	1	4		
10	1 1/2	18		
Check -- Fifteen trees were inoculated in the trunk. One became infected in 221 days.				
Table 2. Root Inoculations, May 4, 1955				
11	1 1/2	5	370	
12	2	8	125	
13	2	7		
14	2	12		
15	3	3		
16	3	6		
17	2 1/2	6		
18	2 1/2	3		
19	1	3		
20	2	6	125	
Check -- 89 trees infected of 93 trunk-inoculated. Check inoculum was spores in distilled water. (20 trees in 30 days; plus 53 trees in 43 days, plus 16 trees in 59 days = 89 wilted.)				
Table 3. Root inoculations, May 16, 1956				
21	1	3 1/2	73	Intermediate
22	1 1/4	6	73	Codominant
23	1 1/2	4		Codominant
24	2	3 1/2		Codominant
25	1 1/4	6 1/2	467	Codominant
26	1 1/2	4 1/4		Codominant
27	3/4	4 2/3		Codominant
28	1 1/4	4 3/4		Codominant
29	2	5 1/2	73	Intermediate
30	1	8	97	Codominant
31	1 1/4	4 1/2		Intermediate
32	1 1/2	6 2/3	73	Codominant
33	1 1/4	7 2/3		Codominant
34	1	5 1/4		Intermediate
35	1	3 1/3	73	Codominant
Check -- 100 trees infected of 100 trunk-inoculated in 28 to 40 days.				
Table 4. Root Inoculations, June 4, 1957				
36	2	5		
37	3/4	3 1/2	52	
38	1 1/2	7		
39	1	6		
40	1 1/2	6		
41	1	4	45	
42	1	6	45	
43	2 1/2	5 1/2		
44	1 1/2	6	52	
45	1	7 1/2		
Check -- Two of three trees inoculated in the trunk showed symptoms in 34 days.				

distance from the trunk.

The range in size of roots inoculated was from 3/4 inch to 3 inches in diameter. Three-fourths of the roots were in the 3/4- to 1 1/2-inch size range, which provided the most successful inoculations.

Each year, a majority of the successfully root-inoculated trees showed symptoms on a common date. The intervals between inoculation and appearance of symptoms, however, varied greatly from year to year. Spring-inoculated trees showed symptoms in 125 days in 1955, 73 days in 1956, and in 45 to 52 days in 1957.

In every series, except 1957 (1957 observations still in progress), a tree or two did not produce symptoms for more than a year after inoculation. This behavior is difficult to explain, but it is rather typical of what occurs in natural wilt areas, where root grafting appears probable. A tree only a short distance from a wilt tree may develop wilt in the same year, a year later, or 2 to 3 years later than its infected neighbor.

Trees inoculated in the autumn frequently do not show marked symptoms until the following spring (4). The long incubation period of the infected check tree shown in Table 1 is evidence of this. The low percentage of successful inoculations in 1954 (Table 1) could be due to the type of inoculum used (malt agar culture), the time of the year inoculated, or poor technique. Fall inoculations usually have been less effective than spring inoculations (1).

In Table 3, crown class of trees root-inoculated is recorded. Of the 15 trees, one tree was dominant, ten were codominant, and four were intermediate. Seven trees became infected, five were codominant and two intermediate. The limited data do not suggest a relationship between crown class and susceptibility to infection.

DISCUSSION

The results of these 4 years of root inoculations do not support the hypothesis that distance alone is the reason for the long intervals of time between deaths of root-grafted oaks in wilt areas. It was expected that increasing the distance between the inoculation point on the root and the trunk of the tree would result in lengthening the interval to development of foliage symptoms. It may be that the differences in distances represented in the experiments were not sufficiently great to show the effects of distance. However, Table 1 lists a tree inoculated only 4 feet from the trunk that did not show infection for 547 days; in Table 2, tree no. 11 inoculated 5 feet from the trunk did not show symptoms for 370 days; tree no. 25 in Table 3, inoculated 6 1/2 feet from the trunk showed signs in 467 days. Other trees inoculated at greater distances from the trunk showed symptoms in much less time. The behavior of the trees with long delayed symptoms may be due to resistance to the fungus in the roots of these trees.

Genetical differences among these seed-propagated trees may account for differing reactions to inoculation. Some trees may provide better conditions for growth and establishment of the fungus, which in turn would lead to more rapid infection and symptom development.

In each yearly trial, there were differences in incubation times between root and trunk inoculations. Since inoculation distance from the trunk does not adequately explain these time differences, other explanations must be sought. Henry and Riber (2) found that ground level inoculations resulted in longer incubation periods than either root or breast height inoculations. Root and stem form a continuous structure. There is, however, a transition region where root and stem meet, and where the various parts of each organ merge with those of the other. Perhaps growth of the fungus is retarded in this area, thus resulting in the longer incubation period from root inoculations.

Long time intervals between deaths of root grafted trees may be the result of interrupted growth of the fungus at the graft sites. Continuity of the structural elements of the two roots may not be complete. The degree of graft success may be an important factor. The size of the roots grafted may also affect the rapidity of infection spread. In addition, if the fungus had to pass through several grafts this might further prolong the time interval.

The percentage of successful root inoculations as compared with trunk inoculations is low. Roots may possess antibiotic properties that protect them against an environment (the soil) favorable to the survival of soil pathogens. This antibiotic property could be present in varying amounts in different individual trees.

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MORTALITY IN WILT INFECTED OAKS

T. W. Jones[1]

Oak wilt infection in trees of the red oak group usually results in death. Trees of the white oak group are generally less severely affected but little evidence of their mortality rate has been available. In 1957, 462 diseased oaks in southeastern Missouri were re-examined. Wilt infection in each of these trees was detected sometime during the period 1952 through 1956 by observation of typical symptoms and isolation of the causal organism, Ceratocystis fagacearum (Bretz) Hunt.

Results of this survey support the accepted concept of high oak wilt virulence in the red oak group. Of the 365 infected red oaks examined, only three were alive in 1957 (Table 1). Foliar wilt symptoms were observed in each of these three trees for the first time in 1956 and were evident again in 1957. All three trees have probably since died.

The mortality data from infected trees of the white oak group give an entirely different picture of oak wilt virulence. Of the 97 infected white oaks examined, only 19 percent were dead. Twenty-six percent were living but displaying wilt symptoms, and 55 percent were alive with no foliar symptoms of the disease. These figures cannot be considered entirely conclusive for the white oak group, because the disease may again become active in some of the symptomless trees. Also, some of the living trees that display disease symptoms may eventually recover. However, the figures do suggest that the mortality rate in wilt infected white oaks is relatively low.

These results indicate that the threat of the wilt disease to one group of oaks is perhaps less than was once supposed. However, they also emphasize the need for further investigation of the persistence of the oak wilt fungus in nonsymptomatic white oaks and the possible importance of such oaks as reservoirs of inoculum for spread of the disease.

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Table 1. Condition during June and July 1957 of trees infected with oak wilt in previous years.

Species	Trees examined	Dead							Living															Total	
		Infection date							Symptoms present							Symptoms absent									
									Infection date							Infection date									
		'52	'53	'54	'55	'56	Total		'52	'53	'54	'55	'56	Total		'52	'53	'54	'55	'56	Total				
	No.	No.	No.	No.	No.	No.	No.	%	No.	No.	No.	No.	No.	No.	%	No.	No.	No.	No.	No.	No.	%	No.	%
Q. velutina	110	0	18	1	41	48	108	98	0	0	0	0	2	2	2	0	0	0	0	0	0	0	2	2
Q. coccinea	238	6	39	0	81	111	237	99	0	0	0	0	1	1	1	0	0	0	0	0	0	0	1	1
Q. marilandica	16	0	0	2	6	8	16	100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Q. rubra	1	1	0	0	0	0	1	100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
All red oaks	365	7	57	3	128	167	362	99	0	0	0	0	3	3	1	0	0	0	0	0	0	0	3	1
Q. alba	71	0	2	0	6	2	10	14	0	0	0	8	10	18	25	0	5	0	17	21	43	61	61	86
Q. stellata	26	0	0	0	6	2	8	31	0	0	0	5	2	7	27	0	0	0	7	4	11	42	18	69
All white oaks	97	0	2	0	12	4	18	19	0	0	0	13	12	25	26	0	5	0	24	25	54	55	79	81
All oaks	462	7	59	3	140	171	380	82	0	0	0	13	15	28	6	0	5	0	24	25	54	12	82	18

NEW OR UNUSUAL RECORDS OF PLANT DISEASE OCCURRENCE

DOWNY MILDEW ON CUCURBITS IN ILLINOIS IN 1957

By G. H. Boewe

During the late summer and fall of 1957, a severe epiphyototic of downy mildew, Pseudoperonospora cubensis (Berk. & Curt.) Rostow., developed on the fall crop of cucumber and squash in southern Illinois. Also, the disease attacked leaves and stems of watermelons in the same area. This is the first record of the disease on cucumber, squash, or watermelon in Illinois. It was found on muskmelon in southern Illinois (Union County) in August of 1929.

On August 30, 1957, a specimen of cucumber from Alexander County suspected of being infected by downy mildew was received from Mr. John Slaton, Assistant Farm Adviser, Mounds, Illinois. Microscopic examination showed the downy mildew fungus fruiting abundantly on the leaves (Plant Disease Situation #48).

On September 9 and 10, a survey was made of cucurbit plantings in Alexander, Pulaski, and Union Counties in southern Illinois. All plantings of cucurbits examined, irrespective of age, variety, or habitat, were infected, including seedling cucumber plants in the two-leaf stage. The cucumber varieties, Ashley, Palmar, Stono, and Marketeer, were examined. Where comparisons could be made, Ashley had less downy mildew than Palmar or Stono. The oldest planting examined (Alexander County) was so severely affected that only one small picking was obtained. The youngest planting, which showed no fruiting of the fungus on leaf lesions at the time of examination, later was reported by the grower "to be wiped out by the disease in spite of four applications of spray" (probably zineb).

Two plantings of squash, variety Prolific, were examined on one farm. Although 100 percent of the plants and 40 percent of the leaves were infected, the grower reported downy mildew did not "knock out" the squash as badly as the cucumber.

Two growers, one in Union and one in Alexander County, reported having watermelons that died before the fruit matured. Microscopic examination of the limited green tissue that could be found in the field revealed downy mildew fruiting on both stems and leaves. It appeared that these plants were killed by downy mildew.

All but one of the growers visited had dusted regularly with captan and lindane. In addition, some had picked diseased leaves from affected plants and removed them from the patch. Growers who changed from captan dust to liquid zineb sprays reported definite benefits in checking the disease.

U. S. Weather Bureau data from Cairo and Anna show that this region had 125 percent more rainfall during April through July than the long term average, and a mean temperature that was slightly (0.8° F) above the long term average. During the period August through October rainfall was deficient by 22.7 percent and the temperature was 2° below normal.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS NATURAL HISTORY SURVEY, URBANA, ILLINOIS

RHIZOCTONIA ROOT ROT ON STORED HORSERADISHES IN CONNECTICUT

By Saul Rich[1]

Locally grown horseradish roots developed a firm, fibrous, ordorless rot in storage. The rotted tissue, which extended deep into the root from wounds, was lemon-yellow, streaked with brown, and abundantly invaded with the mycelium of Rhizoctonia solani. The symptoms were very similar to those described by Kadow and Anderson[2]. This is the first report of the disease from Connecticut.

THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN, CONNECTICUT

[1]Plant Pathologist.

[2]Kadow, K. J., and H. W. Anderson. 1940. A study of horseradish diseases and their control. Univ. of Illinois Bulletin 469: 567-569.

OCCURRENCE OF DATE PALM INFLORESCENCE ROT IN IRAQ

By F. Hussain

According to Hansford[1] an inflorescence rot of the date palm (Phoenix dactylifera L.) has been occurring in Iraq since at least 1938. Owing to lack of facilities he apparently was unable to attribute the rot to a specific organism with certainty. Aside from references by him to a communication with the Mycological Institute at Kew, in which Mauginiella scaettae was stated as the cause of the disease, the exact situation was obscure until the spring and early summer of 1957. At this time a fungus, corresponding to Mauginiella scaettae as described by Cavara[2], was isolated from the spathes, floral axes, and the appendages of several decaying inflorescences. It was again obtained in December of the same year from tissue taken from the adaxial basal side of a frond, and in February 1958 from young inflorescences not yet emerged.

The disease, which is sporadic, destroys the floral parts, with an occassional loss of flowers as high as 90 percent. Although the disease has been observed in a number of localities in the country, it is most prevalent in the vicinity of Basrah near the Persian Gulf. This is attributed to the relatively high humidity in that section. Growers report variation in susceptibility to the disease among the varieties commonly grown but this has not been confirmed experimentally. Further studies are underway to learn the manner of the overwintering of the fungus, and the effectiveness of fungicide applications for control.

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[1]Hansford, C. G. 1949. Report to the Directorate General of Agriculture.

[2]Cavara, F. 1926. Mauginiella scaettae Cav. Nuovo ifomicete parassita della palma datteri di Cireniaca. Bull. Orto Botanico di Napoli 8: 207-10.

ATTENTION, PLEASE!

AMERICAN PHYTOPATHOLOGICAL SOCIETY GOLDEN JUBILEE VOLUME OF SYMPOSIA

The American Phytopathological Society this year is celebrating its 50th Anniversary. In connection with this Golden Jubilee Anniversary a series of outstanding symposia by internationally-known scientists is being presented at the meeting of this Society with the A. I. B. S. in August, 1958.

A book of approximately 1000 pages entitled: "Plant Pathology -- Problems and Progress 1908-1958", containing over 60 symposium papers will be published by the American Phytopathological Society in the winter of 1958. This volume will be of great value to scientists in many fields of interest, as the papers are primarily basic in content and apply to many phases of the biological and physical sciences.

Included in the list of over 70 scientists whose papers will appear in the book are: K. F. Baker, F. C. Bawden, Barry Commoner, S. D. Garrett, J. G. Harrar, J. G. Horsfall, G. W. Keitt, A. G. Lochhead, S. E. A. McCallan, G. L. McNew, Hubert Martin, P. R. Miller, G. B. Sanford, K. M. Smith, W. C. Snyder, E. C. Stakman, W. M. Stanley, J. A. Stevenson, G. J. M. Van der Kerk, J. C. Walker, J. H. Warcup, Robley Williams, and D. W. Wooley.

A number of topics will be covered in this book, which will be an invaluable reference and text book. Included will be significant achievements over the past 50 years and recent advances in the fields of fungicides, viruses, physiology of parasitism, disease resistance, soil microbiology, biology of root disease fungi, nematology, disease epidemiology, and history and development of plant pathology.

"Plant Pathology -- Problems and Progress 1908-1958", a book of basic principles, will be illustrated, bound in regular book binding, and priced at $8.50. Orders should be placed with the American Phytopathological Society, P. O. Drawer 1106, New Haven 4, Conn. Orders are being accepted now; a limited edition will be published. -- George A. Zentmyer, Chairman, Committee on Publicity and Sales of Jubilee Volume, American Phytopathological Society.

ANNOUNCEMENT

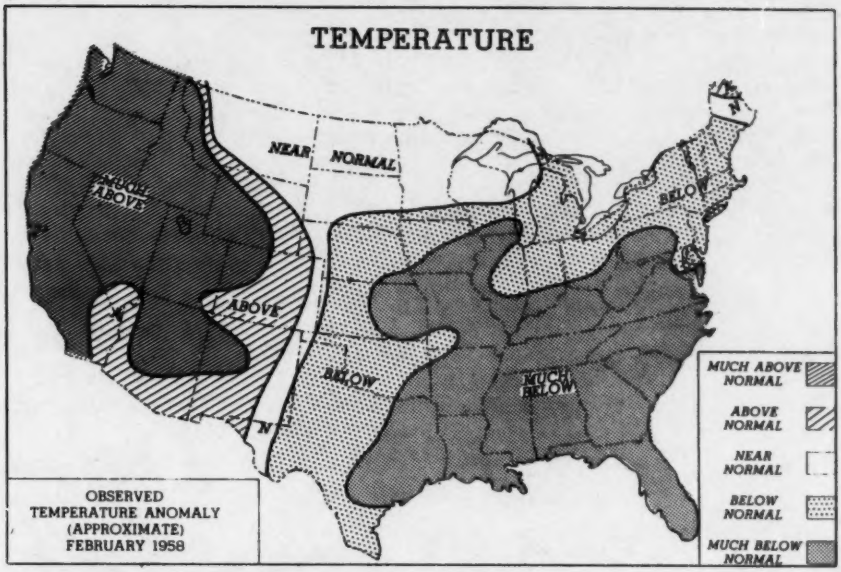
RESULTS OF 1957 FUNGICIDE TESTS

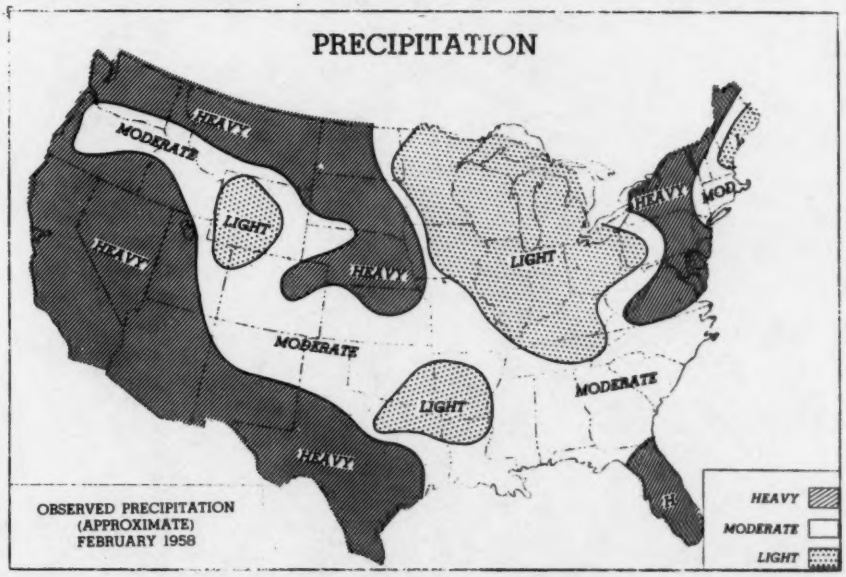
The American Phytopathological Society for several years has sponsored the publication of results from tests on newer fungicides. The "Results of 1957 Fungicide Tests" for the first time has been printed privately and is issued as a single copy. This replaces the previous practice of combining reprints of serial articles published in "Agricultural Chemicals." The "Results of 1957 Fungicide Tests" can be secured at $1.00 per copy only from Dr. A. B. Groves, Department of Plant Pathology and Physiology, Winchester Fruit Research Laboratory, Rural Route 3, Winchester, Virginia. All orders should be accompanied by remittances made out to The American Phytopathological Society. An added charge will be made for postage and handling where orders must be billed.

CORRECTION

REPORTER, February issue: On page 234, the sixth material listed should be "Ortho LM Seed Protectant" (not Prectant).

REPORTER, March issue: On page 318, the title for Figure 1B evidently was partially covered up when the page was photographed. It should read: Shows plants that have died in the field. Note the black roots in both 1A and 1B.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.